TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

THIRTY-FIRST SESSION

HELD AT THE CHALFONTE HOTEL, ATLANTIC CITY, N. J.
JUNE 16, 17 AND 18, 1919

EDITED BY

OSCAR M. SCHLOSS, M.D.

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VOLUME XXXI

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PRESIDENTS

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. William Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. William P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. William D. Booker, M.D.
1902. W. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.
1905. C. G. Jennings, M.D.
1906. A. Jacobi, M.D.
1907. B. K. Rachford, M.D.
1908. C. G. Kerley, M.D.
1909. Charles P. Putnam, M.D.
1910. David L. Edsall, M.D.
1911. Henry Dwight Chapin, M.D.
1912. Walter Lester Carr, M.D.
1913. John Lovett Morse, M.D.
1914. Samuel McC. Hamill, M.D.
1915. George N. Acker, M.D.
1916. Rowland G. Freeman, M.D.
1917. Frank Spooner Churchill, M.D.
1918. Linnaeus E. La Fétra, M.D.
1919. Edwin E. Graham, M.D.

OFFICERS, 1919

President ..... Edwin E. Graham, M.D.
Vice-President ..... Henry Heiman, M.D.
Secretary ..... Howard Childs Carpenter, M.D.
Treasurer ..... Charles Hunter Dunn, M.D.
Recorder and Editor ..... Oscar M. Schloss, M.D.
Member of Committee on Arrangements of the Congress of American Physicians and Surgeons ..... Samuel S. Adams, M.D.
Alternate ..... George H. Acker, M.D.

COUNCIL, 1919

Fritz B. Talbot, M.D., *Chairman*

D. M. Cowie, M.D.
L. Emmett Holt, M.D.
Charles A. Fife, M.D.
Royal S. Haynes, M.D.
Julius P. Sedgwick, M.D.
Isaac A. Abt, M.D.

MEETING PLACES

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3 and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1896. Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.

1902. Boston, May 26, 27 and 28.
1903. Washington, May 12, 13 and 14.
1904. Detroit, May 30, 31 and June 1.
1905. Lake George, N. Y., June 19, 20 and 21.
1906. Atlantic City, N. J., May 30, 31 and June 1.
1907. Washington, May 7, 8 and 9.
1908. Delaware Water Gap, May 25, 26 and 27.
1909. Lenox, Mass., May 27 and 28.
1910. Washington, May 3, 4 and 5.
1911. Lake Mohonk, N. Y., May 31 and June 1 and 2.
1912. Hot Springs, Va., May 29, 30 and 31.
1913. Washington, May 5, 6 and 7.
1914. Stockbridge, Mass., May 26, 27 and 28.
1915. Lakewood, N. J., May 24, 25 and 26.
1916. Washington, May 8, 9 and 10.
1917. White Sulphur Springs, W. Va., May 28, 29 and 30.
1918. Lenox, Mass., May 27, 28 and 29.
1919. Atlantic City, N. J., June 16, 17 and 18.
1920. Highland Park, Ill., May 31, June 1 and 2.

OFFICERS, 1920

President ..... Thomas S. Southworth, M.D.
Vice-President ..... Alfred Hand, Jr., M.D.
Secretary ..... Howard Childs Carpenter, M.D.
Treasurer ..... Charles Hunter Dunn, M.D.
Editor and Recorder ..... Oscar M. Schloss, M.D.

COUNCIL

D. M. Cowie, M.D., *Chairman*

L. Emmett Holt, M.D.
Charles A. Fife, M.D.
Royal S. Haynes, M.D.
Julius P. Sedgwick, M.D.
Isaac A. Abt, M.D.
Richard M. Smith, M.D.

MEMBERS

1903. Abt, Isaac A., M.D. ..... 815 Monroe Building, Chicago
1893. Acker, George N., M.D. ..... 913 Sixteenth Street, Washington
O Adams, Samuel S., M.D. ..... 1801 Connecticut Ave., N.W., Washington
1916. Bartlett, Frederic H., M.D. ..... 44 East Sixty-Third Street, New York
O Blackader, A. D., M.D. ..... 236 Mountain Street, Montreal
1916. Blackfan, Kenneth D., M.D. ..... Johns Hopkins Hospital, Baltimore
1911. Bowditch, Henry I., M.D. ..... 86 Bay State Road, Boston
1918. Brennemann, Joseph, M.D. ..... 5626 Dorchester Avenue, Chicago
1911. Butterworth, Wm. W., M.D. .. 1222 Maison Blanche Bldg., New Orleans
O Caillé, Augustus, M.D. ..... 753 Madison Avenue, New York
1911. Carpenter, Howard C., M.D. ..... 1805 Spruce Street, Philadelphia
O Carr, Walter Lester, M.D. ..... 68 West Fifty-First Street, New York
O Chapin, Henry Dwight, M.D. .... 51 West Fifty-First Street, New York
1897. Churchill, F. S., M.D. ..... 106 Marlborough Street, Boston
1909. Cowie, D. M., M.D. ..... Lawrence Building, Ann Arbor, Mich.
1917. De Buys, L. R., M.D. ..... Maison Blanche Building, New Orleans
1906. Dunn, Charles Hunter, M.D. ..... 178 Marlborough Street, Boston
1904. Eaton, Percival J., M.D. ..... 715 N. Highland Avenue, Pittsburgh

1907. Fife, Charles A., M.D.....2033 Chestnut Street, Philadelphia
1918. Fleischner, E. C., M.D.....350 Post Street, San Francisco
1895. Freeman, Rowland G., M.D..211 West Fifty-Seventh Street, New York
1913. Friedlander, Alfred, M.D..... ...4 West Seventh Street, Cincinnati
1913. Gerstenberger, Henry J., M.D.....1940 Noble Road, Cleveland
1910. Gittings, J. C., M.D.....1805 Pine Street, Philadelphia
1897. Graham, E. E., M.D.....1713 Spruce Street, Philadelphia
1892. Griffith, J. P. Crozer, M.D.....1810 Spruce Street, Philadelphia
1917. Grulee, Clifford, M.D.....104 South Michigan Avenue, Chicago
1898. Hamill, S. McC., M.D.....1822 Spruce Street, Philadelphia
1902. Hand, Alfred, Jr., M.D.....1724 Pine Street, Philadelphia
1910. Haynes, Royal S., M.D.....140 West Fifty-Eighth Street, New York
1909. Heiman, Henry, M.D.....64 West Eighty-Fifth Street, New York
1914. Helmholz, Henry F., M.D.....800 Davis Street, Evanston, Ill.
1910. Herrman, Charles, M.D....250 West Eighty-Eighth Street, New York
1915. Hess, Alfred F., M.D.....16 West Eighty-Sixth Street, New York
O Holt, L. Emmett, M.D.....14 West Fifty-Fifth Street, New York
1913. Hoobler, B. Raymond, M.D.....1563 David Whitney Building, Detroit
1905. Howland, John, M.D.....20 East Eager Street, Baltimore
1918. Jeans, Philip C., M.D.....500 South Kingshighway, St. Louis
1896. Kerley, Charles G., M.D.....132 West Eighty-First Street, New York
1905. Knox, J. H. Mason, Jr., M.D.....“The Severn,” Baltimore
O Koplik, Henry, M.D.....30 East Sixty-Second Street, New York
1903. Ladd, Maynard, M.D.....535 Beacon Street, Boston
1903. La Fétra, Linnaeus E., M.D....113 East Sixty-First Street, New York
1911. Lucas, William Palmer, M.D..University of California, San Francisco
1912. McClanahan, H. M., M.D.....507 McCague Building, Omaha
1909. Machell, H. T., M.D.....459 Avenue Road, Toronto
1917. Marriott, W. McKim, M.D.....Washington University, St. Louis
1898. Miller, D. J. M., M.D..Pacific and California Aves., Atlantic City, N. J.
1918. Mitchell, A. Graeme, M.D.....1717 Pine Street, Philadelphia
1896. Morse, J. Lovett, M.D.....70 Bay State Road, Boston
O Northrup, William P., M.D..61 East Seventy-Third Street, New York
1915. Park, Edwards A., M.D.....Johns Hopkins Hospital, Baltimore
1910. Pisek, Godfrey R., M.D.....26 East Sixty-Fourth Street, New York
1912. Porter, R. Langley, M.D.....240 Stockton Street, San Francisco
1891. Rachford, B. K., M.D.....1961 Madison Road, Cincinnati
1917. Ramsey, Walter R., M.D.....942 Lowry Building, St. Paul, Minn.
1905. Ruhräh, John, M.D.....11 East Chase Street, Baltimore
1900. Saunders, E. W., M.D.....1541 South Grand Avenue, St. Louis
1912. Schloss, Oscar M., M.D.....39 East Sixty-First Street, New York
1917. Schlutz, F. W., M.D.....716 La Salle Building, Minneapolis
1913. Sedgwick, Julius P., M.D.....2015 Kenwood Parkway, Minneapolis
1902. Shaw, Henry L. K., M.D.....361 State Street, Albany
1914. Sherman, DeWitt, H., M.D.....680 West Ferry Street, Buffalo
1915. Smith, Richard M., M.D.....355 Marlborough Street, Boston
1891. Snow, Irving M., M.D..... .....737 Delaware Avenue, Buffalo
1905. Southworth, Thomas S., M.D.....807 Madison Avenue, New York
1911. Talbot, Fritz B., M.D.....311 Beacon Street, Boston
1919. Taylor, Rood, M.D.....614 Syndicate Building, Minneapolis
1914. Van Ingen, Philip, M.D.....125 East Seventy-First Street, New York
1912. Veeder, Borden S., M.D.....608 Humboldt Building, St. Louis
1914. Walls, Frank X., M.D.....1208, 30 North Michigan Avenue, Chicago
1896. Westcott, Thompson S., M.D.....1720 Pine Street, Philadelphia
1913. Wilcox, Herbert B., M.D.....39 East Seventy-Fifth Street, New York
O Winters, J. E., M.D.....25 West Thirty-Seventh Street, New York

Note.—The first column gives the date of election to the Society. The organizers of the Society are designated by “O,” the date being 1888.

HONORARY MEMBERS

Dr. John Thompson.....Edinburgh, Scotland
Dr. George F. Still.....London, England
Dr. William Osler.....Oxford, England
Dr. V. Hutinel.....Paris, France
Dr. Charles Rauchfuss.....Petrograd, Russia
Dr. P. Armand Delille.....Paris, France
Dr. A. Marfan.....Paris, France
Dr. Leonard Findlay .....Glasgow, Scotland
Dr. Truby King.....New Zealand
Dr. E. Weill.....Lyons, France

EMERITUS MEMBERS

Dr. Allen Baines.....Toronto
Dr. Floyd M. Crandall.....New York
Dr. John Dorning.....New York
Dr. Francis Huber.....New York

Deceased

John A. Jeffries, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

Thomas F. Sherman, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

John M. Keating, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

Charles Warrington Earle, M.D.
Born, 1845,
Died, November 19, 1893.

J. Lewis Smith, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

Joseph O'Dwyer, M.D.
Born, October 12, 1841,
Died, January 7, 1898.

John Henry Fruitnight, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

Frederick A. Packard, M.D.
Born, November 17, 1862,
Died, November 1, 1902.

Walter S. Christopher, M.D.
Born, 1859,
Died, March 2, 1905.

Leroy Milton Yale, M.D.
Born, February 12, 1841,
Died, September 12, 1906.

James Park West, M.D.
Born, June 27, 1858,
Died, June 25, 1908.

Frederick Forchheimer, M.D.
Born, 1853,
Died, June 1, 1913.

Thomas Morgan Rotch, M.D.
Born, 1850,
Died, March 9, 1914.

Charles P. Putnam, M.D.
Born, 1845,
Died, April 22, 1914.

Henry Leber Coit, M.D.
Born, 1854,
Died, March 13, 1917.

A. Jacobi, M.D.
Born, May 6, 1830,
Died, July 10, 1919.

CONSTITUTION AND BY-LAWS OF THE AMERICAN PEDIATRIC SOCIETY

ARTICLE I

Name and Object of the Society

The Society shall be known as the American Pediatic Society, and shall hold an annual meeting.

It has for its object the advancement of the knowledge of physiology, pathology and therapeutics of infancy and childhood.

ARTICLE II

Proceedings

The proceedings shall consist of:

1. Discussions on subjects previously selected.
2. Original communications.
3. Demonstrations of gross and microscopic preparations, apparatus, and instruments.

ARTICLE III

Members

The Society shall be composed of three classes of members to be designated: (1) active members; (2) emeritus members, and (3) honorary members.

The number of active members shall be limited to seventy-five.

The number of emeritus members shall be unlimited.

The number of honorary members shall be limited to twenty-five.

ARTICLE IV

Election of Active Members

Nominations to membership, signed by two members of the Society, must be made in writing at least one meeting prior to election.

Nomination should be made to the Secretary, whose duty it shall be to require the nominators to write a personal letter endorsing and stating the qualifications of the nominee, and at the same time furnishing a list of the nominee's professional position and publications, with reprints of the latter, when obtainable.

It shall be the duty of the Secretary to transmit to the Chairman of the Council all the above papers pertaining to each nominee, at least three months prior to his possible election.*

The Chairman of the Council shall transmit the above-named papers to the other members of the Council in the order of their seniority as Council members, the junior members of the Council returning them to the Secretary.

It shall also be the duty of the Secretary to furnish a printed list of all nominees at least once a year to every member of the Society, and it shall be the duty of the members of the Society, without solicitation, to furnish the Council with any information that they may possess as to the fitness of the nominees to become members of the Society.

On nomination by the Council, members shall be elected by the Society by ballot. A two-thirds vote of the members present shall be necessary for election.

ARTICLE V

Election of Emeritus and Honorary Members

Active members who have served fifteen years and have paid all their dues, may on their request and on the recommendation of the Council be made emeritus members. They shall be entitled to attend meetings and read papers, when they so desire, but cannot vote.

Physicians of sufficient eminence to merit the distinction may be elected honorary members, to a number not exceeding twenty-five. Such members shall be entitled to attend all meetings and take part in the proceedings, but cannot vote. Honorary members shall be elected in the same way as active members.

ARTICLE VI

Initiation Fee and Annual Dues

Election to membership shall be completed by the payment of an initiation fee of $10.

Each active member shall pay an annual fee, the amount of which shall be decided at each annual meeting.

Emeritus and honorary members shall be exempt from fees.

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* In the event that required information relating to candidates for membership is not furnished to the Secretary by three months before the time of the meeting at which they would be considered, such candidates for election shall be held over for another year. (Resolution adopted 1912.)

ARTICLE VII

Officers

The officers shall consist of a President, Vice-President, Secretary, Recorder [and Editor], Treasurer and a Delegate to the Congress of Physicians and Surgeons.

The officers shall be elected annually on nomination by the Council.

ARTICLE VIII

Duties of Officers

The duties of the President, Vice-President, Secretary and Treasurer shall be those usual to these offices. The Recorder [and Editor] shall secure the papers read and see that proper notes are taken of the discussion thereon for the use of the committee on publication.

ARTICLE IX

The Council

The Council shall consist of seven members, the senior member being Chairman. One member of the Council shall be elected each year. Four members shall constitute a quorum. The senior member shall retire at the end of each year and shall not be immediately eligible to reelection.

ARTICLE X

Duties of Council

The duties of the Council shall be to consider nominations for membership and report them to the Society. The votes of four members of the Council shall be required for nomination. The Council shall also nominate the officers of the Society and shall decide the time and place of meeting.

ARTICLE XI

Committee of Arrangements

The President, Secretary and the Chairman of the Council shall constitute a committee of arrangements, the President being chairman of this committee. They shall arrange the details of the meeting and the preparation of the program, and they shall have the authority to invite guests to attend the meeting and to participate in the discussion.

ARTICLE XII

Publication Committee

The Secretary, Treasurer and Recorder shall constitute a committee on publication, to which shall be referred all papers, reports and other matters intended for publication.

All papers presented shall become the property of the Society.

ARTICLE XIII

Amendments of Constitution and By-Laws

Proposals for amendments of the constitution and by-laws must have been made at the meeting previous to that at which they are voted on, the notice of which shall contain an announcement of the proposed changes. Such changes shall require, for their adoption, an affirmative vote of three-fourths of the active members present.

ARTICLE XIV

Termination of Membership

A member may be expelled from the Society for conduct unbecoming a physician and a gentleman. In such cases, formal charges must be made in writing by two members, which shall be referred to the Council.

Active membership shall lapse for any one of the following reasons:

(1) Absence from three consecutive meetings without excuse acceptable to the Council; (2) failure to present and read a paper for five consecutive years; the Secretary in both these cases shall notify members one year before date of possible lapse; (3) non-payment of dues for two years, two notifications having been sent by the Treasurer.

ARTICLE XV

Quorum

Any number of members present at the appointed time of the annual meeting shall constitute a quorum for the transaction of ordinary business, but for the election of members, fifteen shall be necessary for a quorum; and for the expulsion of members, or for altering the constitution and by-laws, twenty-five members shall be necessary.

ARTICLE XVI

Order of Business

1. The President shall call the meeting to order and deliver an annual address. In his absence the Vice-President shall preside, and in the absence of these officers, the Chairman of the Council.

2. When a general discussion is arranged by the Council, the two members appointed to open the discussion shall not occupy more than twenty minutes each; subsequent speakers shall be restricted to ten minutes each.

3. Papers shall not exceed fifteen minutes in the reading. In the discussion following the reading of such papers, remarks shall be limited to five minutes. Should any paper be too long to read in fifteen minutes, the writer must prepare an abstract which can be read within that time.*

4. At the business session the report of the Council as a committee on nominations to office and to membership, shall be made, and ballot shall be held thereon.

Adopted May 28, 1900.

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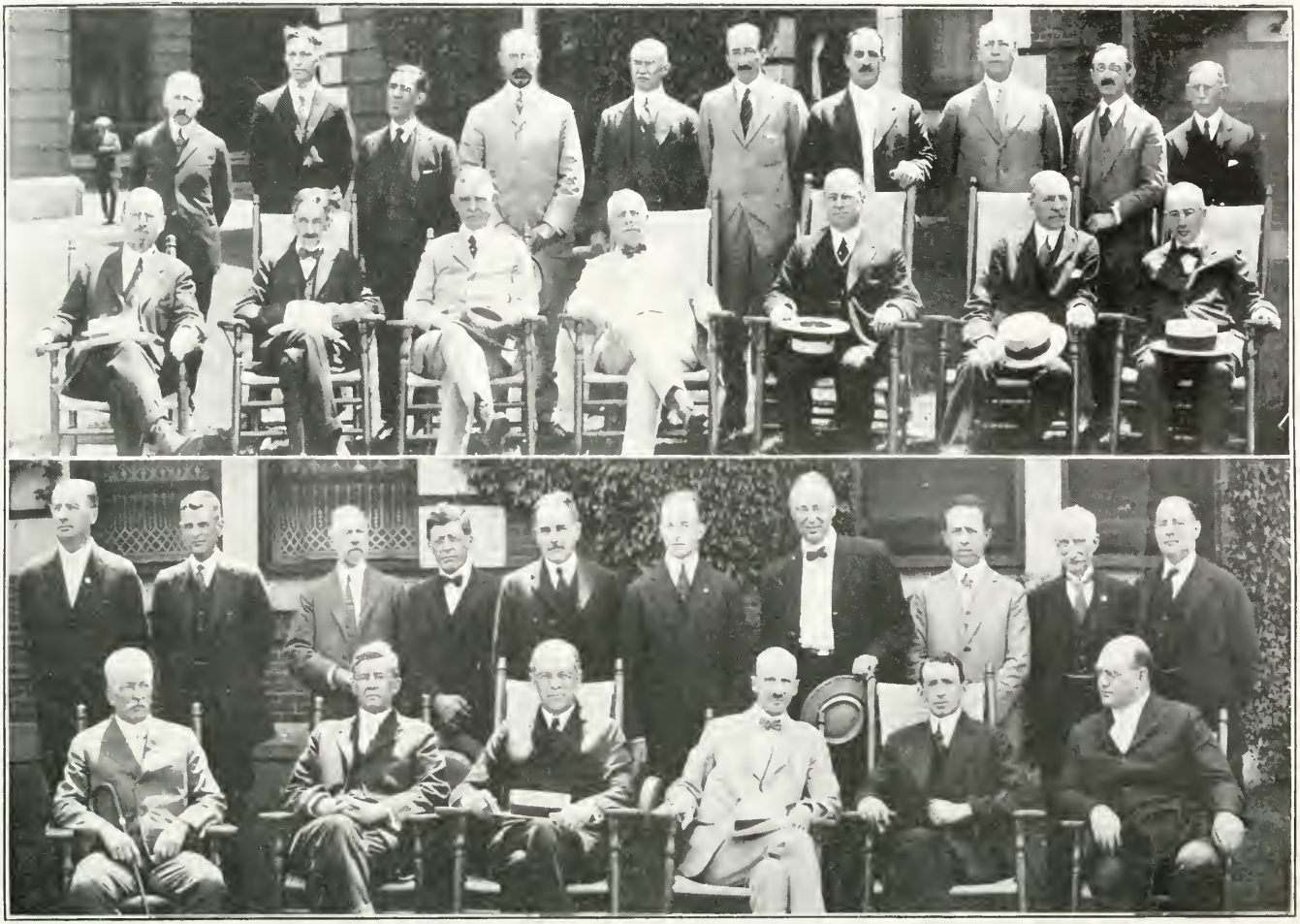
*In order to expedite the publication of the Transactions, all members whose papers are to appear in the volume, shall be required either to have their articles in the hands of the editor before July 1 or to have them in process of publication in some medical journal by that date. In the latter event, the editor of the Transactions is to be notified what journal will publish the paper and reprints or galley proofs are to be furnished to him as soon as possible. (Resolution adopted, 1912.)



MEMBERS OF THE AMERICAN PEDIATRIC SOCIETY AT ATLANTIC CITY MEETING, 1919.

MINUTES OF THE THIRTY-FIRST ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY

Held at the Chalfonte Hotel, Atlantic City, N. J.
June 16, 17 and 18, 1919

The meeting was called to order at 10 a. m. by the President, Dr Edwin E. Graham of Philadelphia.

The following members were present during the sessions: Drs. Isaac A. Abt, Chicago; George N. Acker, Washington; Samuel S. Adams, Washington; K. D. Blackfan, Baltimore; Joseph Brennemann, Chicago; Howard Childs Carpenter, Philadelphia; Walter Lester Carr, New York; Henry Dwight Chapin, New York; D. Murray Cowie, Ann Arbor; L. R. DeBuys, New Orleans; Percival J. Eaton, Pittsburgh; Charles A. Fife, Philadelphia; E. C. Fleischner, San Francisco; Rowland G. Freeman, New York; J. Claxton Gittings, Philadelphia; E. E. Graham, Philadelphia; J. P. Crozer Griffith, Philadelphia; S. McC. Hamill, Philadelphia; Alfred Hand, Jr., Philadelphia; Henry Heiman, New York; Henry F. Helmholz, Evanston, Ill.; Charles Herrman, New York; L. Emmett Holt, New York; P. C. Jeans, St. Louis; Charles G. Kerley, New York; J. H. M. Knox, Jr., Baltimore; Henry Koplik, New York; L. E. La Fétra, New York; H. M. McClanahan, Omaha; W. McKim Marriott, St. Louis; H. T. Machell, Toronto; D. J. Milton Miller, Atlantic City; A. Graeme Mitchell, Philadelphia; J. Lovett Morse, Boston; Edwards A. Park, Baltimore; Godfrey R. Pisek, New York; R. Langley Porter, San Francisco; B. K. Rachford, Cincinnati; Walter R. Ramsey, St. Paul; John Ruhräh, Baltimore; Oscar M. Schloss, New York; Frederic W. Schlutz, Minneapolis; Julius P. Sedgwick, Minneapolis; Henry L. K. Shaw, Albany; DeWitt H. Sherman, Buffalo; Richard M. Smith, Boston; Thos. S. Southworth, New York; Fritz B. Talbot, Boston; Phillip Van Ingen, New York; Thompson S. Westcott, Philadelphia; Herbert B. Wilcox, New York, and J. E. Winters, New York.

In addition to the members, the following guests were introduced: Sir Arthur Newsholme, England; Drs. Walter W. Strang, New York; H. L. Dwyer, New York; Anna E. Rude, Washington, D. C.; C. K. Johnson, Burlington, Vt.; E. V. Milholland, Baltimore; L. W. Sauer, Evanston, Ill.; A. J. Scott, Los Angeles; J. Gurney Taylor, Milwaukee; Fred Moore, Des Moines; Frank Gegenbach, Denver; J. H. E. Resa-

mond, Memphis, Tenn.; Arthur A. Shawkey, Charleston, W. Va.; Lewis W. Elias, Asheville, N. C.; J. D. Love, Jacksonville, Fla.; M. B. Herlong, Jacksonville, Fla.; Percival J. Nicholson, Ardmore, Pa.; Horace H. Jenks, Philadelphia; John A. Card, Poughkeepsie, N. Y.; H. J. Cartin, Johnstown, Pa.; Edith Brownson, San Francisco; C. A. Sparrow, Worcester, Mass.

It was moved, seconded and carried that the reading of the minutes of the last meeting be dispensed with.

The following papers were presented:

1. Dr Edwin E. Graham, Presidential Address, on the subject, "Foreign Bodies in the Food and Air Passages."

2. Dr. Fritz B. Talbot: "The Chemistry of Human Milk."

The paper was discussed by Drs. Holt, Sherman and Talbot.

3. Dr. W. McKim Marriott: "The Pathogenesis of Certain Nutritional Disorders."

The paper was discussed by Drs. Schloss, Abt and Marriott.

4. Dr. John Lovett Morse: "A Study of the Relationship of Convulsions in Infancy and Childhood to Epilepsy."

The paper was discussed by Drs. Koplik, DeBuys, Griffith, Heiman, Holt, Freeman, Adams, Shaw, Helmholz, Talbot and Morse.

5. Dr. E. C. Fleischner and Dr. Karl F. Meyer: "Certain Aspects of Cutaneous Hypersensitiveness."

The paper was discussed by Drs. Cowie and Fleischner.

6. Dr. J. Claxton Gittings: "Quarantine and Disinfection in Scarlet Fever."

The paper was discusssed by Drs. Herrman, Koplik, Miller, Griffith, Heiman, Knox and Gittings.

7. Dr. K. D. Blackfan and Dr. J. L. Gamble: "A Study of the Cholesterol Metabolism of Infants."

8. Dr. Langley Porter: "Certain Nutritional Disorders in Infants Associated with a Proteolytic Intestinal Flora."

The paper was discussed by Drs. Koplik, Fleischner and Porter.

9. Dr. Henry Koplik: "Transfusion in Infants and Children — the Indications and Dangers."

10. Dr. Fritz B. Talbot: "The Energy Metabolism of Children from Birth to Puberty."

The paper was discussed by Drs. Holt and Talbot.

11. Dr. Henry Heiman: "Polioencephalitis Following Influenza During the Recent Epidemic."

The paper was discussed by Drs. Ruhräh, Koplik, Abt, Smith, Talbot and Heiman.

12. Dr. Henry L. K. Shaw: "The Cause of a 'Sporadic' Case of Cerebrospinal Meningitis."

The paper was discussed by Drs. Talbot, Holt, Koplik and Heiman.

13. Dr. Alfred Hand, Jr.: "The Tertiary Cerebral Manifestations of Hereditary Syphilis."

14. Dr. P. C. Jeans: "Cerebrospinal Involvement in Hereditary Syphilis."

The paper was discussed by Drs. Marriott, DeBuys, Abt and Jeans.

15. Dr. Richard M. Smith: "A Health Study of a Boys' School."

The paper was discussed by Drs. Talbot, Hamill, Southworth, La Fétra, Abt, Helmholz, Shaw, Holt, Heiman and Smith.

16. Dr. Thomas S. Southworth: "The Predominance of Seborrheic Eczema in Early Life."

17. Dr. Fritz B. Talbot: "Report of War Emergency Child Welfare Committee."

18. Dr. Henry Heiman: "A Case of Aneurysm of the Ascending Arch of the Aorta in a Boy of Thirteen Years."

19. On the motion of the Society Dr. Heiman presented a second case: "A Case of Typhoid Fever Infected from Mother's Milk."

This case was discussed by Drs. Smith and Heiman.

20. Dr. Isaac A. Abt: "Some Details in the Management of a Premature Baby." (Lantern demonstration.)

21. Dr. L. Emmett Holt: "Experiences at the Red Cross Conference at Cannes." (Lantern demonstration.)

22. Dr. John Ruhräh: "An Interesting Lung Condition with Exhibition of X-Ray Plates."

This case was discussed by Drs. Heiman and Ruhräh.

23. Dr. Percival J. Eaton: "A Case of Calculus and a Case of Scorbutic Hematuria."

24. Dr. L. R. DeBuys: "A Case of Congenital Anomaly of the Diaphragm with Gastric and Intestinal Obstructions." (Illustrated.)

25. Dr. Howard Childs Carpenter: "A Case of Trichinosis."

This case was discussed by Drs. Abt and Carpenter.

26. Dr. Rowland G. Freeman: "Two Cases of Pyelitis and a Case of Suppression of Urine for Four Days."

This case was discussed by Drs. Smith, Pisek and Freeman.

27. Dr. Charles Gilmore Kerley: "A Few Roentgenograms Demonstrating Gastrointestinal Abnormalities."

This paper was discussed by Drs. McClanahan, Sherman, Pisek and Kerley.

28. Dr. E. A. Park and Dr. G. F. Powers: "Acrocephaly with Deformities of the Extremities."

The paper was discussed by Drs. Herrman and Park.

29. Dr. Joseph Brenneman: "The Incidence and Significance of the Rheumatic Nodules in Children."

The paper was discussed by Drs. Heiman and Brenneman.

30. Dr. H. M. McClanahan: "Osteogenesis Imperfecta."

The paper was discussed by Drs. Marriott, Brenneman, Holt, Herrman and McClanahan.

31. Dr. Joseph E. Winters: "The Care and Treatment of Whooping Cough Patients."

The paper was discussed by Drs. Adams and Winters.

32. Dr. Henry Dwight Chapin: "Do Calories Measure the True Value of Foods?"

The paper was discussed by Drs. Talbot, Sedgwick, Schlutz, Marriott and Chapin.

33. Dr. L. R. DeBuys: "Study of the Stools of Institutional Children to Determine the Existence of Intestinal Parasitic Infection."

The paper was discussed by Drs. Sedgwick, Brenneman, Schloss, Carpenter, Marriott, Mitchell, Park and DeBuys.

34. Dr. Henry F. Helmholz and Dr. L. W. Sauer: "Sugar Tolerance in Atrophic Infants."

The paper was discussed by Drs. Schloss, Marriott, Talbot and Helmholz.

35. Dr. Langley Porter: "Case of Hodgkin's Disease in a Girl of Two Years." Read by title.

EXECUTIVE SESSION

The report of the Council was read by the Chairman of the Council, Dr. Fritz B. Talbot, as follows:

The Council recommended that the following members be excused for absence from this meeting: Drs. Blackader, Grulee and Veeder.

The Council recommended that the resignation of Dr. Wilder Tileston be accepted with regret.

The Council presented the following nominations for officers: President, Dr. Thomas S. Southworth, New York; Vice-President, Dr. Alfred Hand, Jr., Philadelphia; Secretary, Dr. Howard Childs Carpenter, Philadelphia; Treasurer, Dr. Charles Hunter Dunn, Boston; Editor and Recorder, Dr. Oscar M. Schloss; Member of Council, Dr. Richard M. Smith, Boston.

The Treasurer's report was received, examined by an auditing committee and found correct.

The Council recommended that the annual assessment remain at $15.

The Council recommended for election to active membership Dr. Rood Taylor, Rochester, Minn.; for honorary membership: Dr. P. Armand Delille, Paris; Dr. A. Marfan, Paris; Dr. Leonard Findlay, Glasgow; Dr. Truby King, New Zealand; Dr. E. Weill, Lyons.

The Council recommended that the Transactions of the 1919 meeting be published by the American Medical Association.

It was decided that the next meeting should be held at the Moraine Hotel, Highland Park, Ill., the time of the meeting to be May 31 and June 1 and 2.

The Council recommended the appointment of a committee on Child Hygiene to represent the American Pediatric Society, and that Dr. Richard M. Smith be nominated as chairman, with power to appoint the other members of his committee. One of the functions of this committee shall be to outline a course on child hygiene appropriate for introduction into the curriculum of medical schools, and to present said outline for the approval of the Council.

The following letter was presented from the Chief of the Federal Children's Bureau:

To the Chief Executive Council, American Pediatric Association.

My Dear Dr. Talbot:—The Children's Bureau would very much value the services if the Pediatric Association could feel justified in designating a mem-

ber to serve on a medical committee advisory to the Hygiene Division of the Bureau.

Hoping that this cooperation can be arranged, I am

Yours very sincerely,

[Signed] JULIA C. LATHROP.

The Council recommended that Dr. Richard M. Smith, Chairman of the new Committee on Child Hygiene, be appointed as the representative of this Society on the Medical Advisory Committee of the Child Hygiene Division of the Federal Children's Bureau.

The Council recommended the following resolution:

Resolved, That the American Pediatric Society at its Thirty-First Annual Meeting on June 18, 1919, petitions the American Red Cross Society to give due consideration in the formulation of its working program to the subject of child hygiene in this country.

HOWARD CHILDS CARPENTER, M.D., *Secretary*.
OSCAR M. SCHLOSS, M.D., *Recorder*.

REPORT OF WAR EMERGENCY CHILD WELFARE COMMITTEE

Read by the Chairman, Dr. Fritz B. Talbot

Until the late fall of 1918, two members of your Committee continued to meet with the Child Welfare Committee of the Council of National Defense, but up to this time no actual progress had been made in solving the problem of child welfare which confronted the nation.

On Dec. 2, 1918, Surgeon-General Blue called to Washington a group of pediatrists which included four members of your Committee, to consider how the United States Public Health Service should undertake to handle the problem of child welfare. It was brought to their attention that under the executive order of July 1, 1918, the United States Public Health Service was placed in charge of all the public health activities of this country. Since child welfare is a fundamental part of public health work, the United States Public Health Service decided to outline plans for its development. A program on child conservation was drawn up, the introduction to which was as follows:

"No program of child conservation will be satisfactory that does not take into consideration the different requirements of children at various age periods. In other words such a program must include provisions for:

"1. Safeguarding the health of expectant mothers.

"2. Improving the character of the obstetrical and nursing care and lying-in facilities during child birth.

"3. Securing the accurate registration of all births.

"4. Controlling conditions harmful to the health of infants.

"5. Supervising the health of the child and his environment during the preschool and school age.

"6. Safeguarding the health of young children in industry.

"A summary of the general character of the activities to be carried on under such a program is given herewith. Many of them are already being carried on by the Public Health Service; in practically all of these, the Public Health Service is ready to continue and extend its operations in cooperation with

federal, state and local health authorities and with other recognized public and private organizations."

Several individual conferences were held and so far as we are able to determine nothing as yet has been undertaken by the United States Public Health Service because of lack of money.

In the end of March, 1919, three of your committee, Drs. Holt, Hamill and Talbot, sailed for France, at the invitation of the Inter-Allied League of American Red Cross Societies to attend an international conference at Cannes. The history of this conference is briefly as follows:

The call was issued at the request of the League of Nations, the Red Cross societies of the United States, France, Great Britain, Italy and Japan, to meet and "to formulate and to propose to the Red Cross societies of the world an extended program of Red Cross activities in the interest of humanity." The conference opened at Cannes, April 1, 1919, at the call of the Inter-Allied League of Red Cross Societies.

Under Article XXV of the covenant of the League of Nations, members of the League "agree to encourage and promote the establishment and cooperation of duly authorized voluntary national Red Cross organizations, having for their purpose the improvement of health, the prevention of disease and the mitigation of suffering."

Professor Roux, the director of the Institute of Paris, was elected chairman of the conference and Dr. L. Emmett Holt secretary. The conference was subdivided into the following sections:

Dr. Welch, Chairman of the Executive Committee.
Dr. Biggs, Chairman of the section on preventive medicine.
Sir Arthur Newsholme, Chairman of Child Welfare.
Dr. Calmette, Chairman of Tuberculosis.
Dr. Laveran, Chairman of Malaria.
Dr. Ducrey, Chairman of Venereal Disease.
Col. Snow, Chairman of Publication, Education and Statistics.
Miss Stimson, Chairman on Nursing.

The interest of the American Pediatric Society naturally centers on child welfare. The report of the Child Welfare Committee, which was adopted by the conference, was as follows:

CHILD WELFARE COMMITTEE REPORT FROM INTERNATIONAL CONGRESS

Child welfare is an essential part of a general program in public health and preventive medicine and cannot be separated from it. It is particularly closely associated with housing and food production and supply, for children require food not merely for maintenance but for growth.

Children as the weakest members of the community feel first and feel most heavily the effect of all unfavorable hygienic, social and economic conditions. The war has increased infant and child mortality and in some countries the birth rate has been lowered more than one half. The health of many children who have survived has become greatly impaired by reason of improper food and neglect. Furthermore, the war and recent epidemics have enormously increased the number of dependent children, especially in the devastated countries.

A reduced birth rate and an abnormal sickness and death rate may be expected to continue for several years to come; a reduced birth rate owing to death of potential fathers, to social and economic conditions which have affected

the health of potential mothers, and to many other factors; an increased sickness and death rate because of the scarcity and high cost of food, and general hardship and privation which favors the spread of epidemics and lower resistance to all forms of disease. A world wide child welfare campaign is, therefore, one of the most urgent needs of the day, both from a humanitarian and economic standpoint; as the children are the nation's greatest asset.

The pressing problems now are:

1. To save the infants yet to be born and to promote their healthy development.

2. To restore the health and make possible the normal growth and development of children who are now suffering from disease or defective nutrition and to safeguard the health of those whose nutrition has not yet suffered.

3. To do something for the immediate needs of dependent children.

Experience has shown that no efforts in public health work produce more immediate or far-reaching results than those which are put forth in child welfare.

The work relating to child welfare may be divided into six parts, corresponding to the period of child life to which it is directed:

1. *Eugenic consideration affecting the prospective parents.*

2. *The prenatal period in which the child is saved through care of the mother.* The importance of this may be appreciated by the fact that even in the highly civilized countries in peace times (*a*) the still births are equal to between 4 and 5 per cent. of the living births; (*b*) that as a result of premature birth many infants born die in the first weeks of life; (*c*) that other causes of the high mortality during the early weeks are due to conditions affecting the mother.

Methods of prenatal care will necessarily differ in urban and rural communities and also will be modified by race, custom and environment. The essential features must be the supervision and education of the expectant mother by trained public health nurse, midwife, or health visitor from an early period of her pregnancy, aided when possible by the advice and assistance of a physician. The work is greatly facilitated by voluntary registration of pregnant women, which should be encouraged everywhere and it should be closely connected with maternity and child welfare activities.

Success in any effort in child welfare is largely dependent on the intelligent cooperation of both parents; no plan of education, therefore, is complete which does not include the father. This is particularly important in the prenatal period of infancy.

3. *Obstetrical care,* which should include the services of a nurse as well as those of a physician or midwife. Proper care saves the child from accidents during delivery; saves the mother from immediate and remote dangers incident to her confinement; greatly increases the chances of successful breast feeding and prevents much blindness.

4. *Birth to school age.*—This period is sometimes divided into infancy, including the first year or possibly two years, and a preschool age, extending from this time up to 5 or sometimes 6 years.

The important concern during the first two years is the child's nutrition; and the essential thing is that the child be kept under continuous observation and supervision. Everything possible should be done to promote breast feeding, as by far the most important means of insuring vigorous growth and development and preventing infant mortality. At first, the supervision must be

close, the infant being seen in many cases every week or even oftener. The visits will be less frequent as the child grows older, being made at least monthly after six months, and every two months up to the end of the second year. Success during this period depends almost entirely on our ability to educate the mother and to arouse and maintain her interest in her child's growth and progress.

The principal methods are: (*a*) group instructions and individual advice in a central place known as a "milk station," a "child welfare station," a "consultation," etc., and (*b*) home visiting by public health nurses or health visitors. Both methods have their place and should usually be combined certainly in urban communities.

The problem of infancy up to the school period is much the same and can be carried on with the same organization. A quarterly visit to the home or by the child to the central stations and a full medical examination once a year should be the minimum. Much better results would be obtained if the observations were made more frequently. The above requirements should be for every healthy child. Those who are delicate or ill should have special medical attention.

By these means, the beginnings of organic disease may be detected, physical defects and deformities prevented or corrected, and a general supervision maintained over the child's diet and general hygiene.

Records should be kept of all abnormal conditions and of any serious illness, also of height and weight, which will in themselves furnish a good guide as to health progress.

Permanent institutional care for infants and young children should be discouraged on account of the almost insuperable difficulties in maintaining nutrition in infancy under these conditions, and because of the great susceptibility of young children to infection, preference should be given to placing such children in suitable families. All crèches, day nurseries and the like should be under close medical attention.

3. *In the school period.*—At the age of from 6 to 14 or 16 years, the problem is to secure normal growth and physical and moral development, to recognize and correct defects which interefere with these, and to reduce to the minimum the risks of contagious disease. The school physician, the school nurse and the school teacher all have important functions. The school, furthermore, offers an opportunity the value of which is only beginning to be appreciated, to interest, instruct and train the child himself in health matters. It should be the duty of schools to teach health as much as to teach reading and writing. Health work for the schoolchild is inextricably bound up with the problems of general education, and all teachers should receive training which would qualify them to teach the simple facts of health and personal hygiene.

An important kind of health education which can be carried on in the schools is the instruction of girls from the age of 10 years upward in the rudiments of infant care. This has been proved to be valuable in preparing them to undertake the responsible charge of their younger brothers and sisters and what is even more important in assisting to prepare them for the future duties of motherhood.

The child's mind is virgin soil for health instruction of every sort. It is not necessary at this period to clear away the rubbish of prejudice, superstition and habits based on ignorance, which make health education so difficult and often so futile in adult life. Scientific facts in regard to food, health habits, hygiene and the spread and control of disease can, if presented by writers and

artists who know how to teach the child's mind, be made both interesting and attractive. Why should not the stories of the conquest of smallpox, malaria and yellow fever be made as thrilling as the conquests of Alexander or Napoleon? This instruction should be begun with children of 8 or 9 for early impressions are the most lasting. The bare facts of hygiene or physiology as presented in school text-books make little impression that is permanent. Their acquisition is made work. Health instruction should and can be made play; health may even be made a competitive game. If the schoolchildren can be properly instructed in health matters we need have no anxiety about the adults of the next generation.

Progress in the pupil's health is best shown by the weight record; thus an incentive can be furnished for the observance of health rules which are taught.

Health records, at least a record of weight and growth, should be kept during school life. In an ideal scheme, a medical examination should be made once a year of every school child and those who are found to be physically below normal or who do not make a normal gain in weight should receive special attention and supervision during their school life.

6. *Industrial period, 14 to 18 years.*—In connection with legislation relating to child labor, the following fundamental principles should be embodied: (*a*) No child should be allowed to enter industry without first passing a physical examination by a competent physician, showing that he is able to perform the work intended. (*b*) No child should be permitted to continue in any form of work which prevents his normal growth and physical development as determined by accepted standards, and periodic examinations should be made up to the age of 18 years.

Any intelligent action must be based upon an exact knowledge of conditions; therefore, the first step should be a survey. After a survey has been made in any activities which are contemplated there are certain fundamental principles which should be kept in mind if the work is to be effective and anything permanent accomplished.

1. Local customs, climatic conditions, diet and other things upon which are based the habits of the people should be carefully considered and appreciated.

2. All local health habits and racial customs which are good should be accepted and used as a foundation on which to build.

3. It is impossible to formulate beforehand a plan of procedure which can be closely followed. Any plan adopted must have great elasticity and be modified by circumstances as they arise in the course of the work.

4. Local agencies, organizations and individual workers should be developed and utilized so far as possible.

5. The utmost tact is necessary to adapt knowledge and experience gained elsewhere to work which is instituted under new conditions and among new people.

6. No plan of work should be made so complex or so expensive that it cannot be duplicated or extended by a simple organization elsewhere.

7. Success depends much less upon the program adopted than upon the personnel which will put it into effect.

8. To carry out effectively any scheme of child welfare such as has been outlined, it will be necessary to provide material assistance in a considerable proportion of cases.

It was moved and carried that the report be adopted.

FOREIGN BODIES IN THE AIR AND FOOD PASSAGES *

EDWIN E. GRAHAM, M.D.
PHILADELPHIA

During the past two years, through the courtesy of Dr. Chevalier Jackson, I have had an opportunity of seeing in children a comparatively large number of cases of foreign bodies in the air passages, and a much smaller number of cases of foreign bodies in the food passages. I feel much indebted to Dr. Jackson for the opportunity of studying these cases, as all of them were referred to him, and he later referred them to me for medical examination as to the location of the foreign body and for medical treatment.

The subject has been of such great interest to me, and has taught me so much that I feel it may interest you if I draw attention to some of the points that seem to me most important in connection with these foreign body cases.

Of the comparatively large number of cases that I have been seeing, I have selected some of the most interesting. A brief summary of the clinical history of four of these cases will suffice to illustrate their nature.

REPORT OF CASES

Case 1.—James W. F., aged 4 years, was admitted to Jefferson Hospital, April 19, 1919. Diagnosis: Carpet tack in right lung. Location of foreign body: lower lobe of right lung. Tack aspirated, March 15, 1919. Two previous unsuccessful bronchoscopies. Foreign body removed by Dr. Chevalier Jackson, April 23, 1919. Time: thirty-five minutes, ten seconds. Uneventful recovery. Discharged, April 26 (Fig. 1).

Case 2.—Frank S., aged 2 years, was admitted to Jefferson Hospital, March 19, 1919. Diagnosis: Peanut kernal in right bronchus. Location of foreign body: right bronchus at orifice of upper lobe bronchus. Peanut aspirated, March 16, 1919. Symptoms: Marked wheezing, cyanosis, severe cough, fever. Tracheotomy: March 20, 1919. Kernel removed by Dr. Chevalier Jackson, March 25. Time: four minutes, twenty-six seconds. Recovery (Fig. 2).

Case 3.—William B., aged 4 years, admitted to Jefferson Hospital, Dec. 20, 1917. Diagnosis: Staple in left bronchus. Location of foreign body: in left main bronchus. Staple aspirated, Dec. 14, 1917. Foreign body removed by Dr. Chevalier Jackson, Jan. 8, 1918. Time: eighteen minutes, fifty seconds. Recovery. Discharged, January 11 (Fig. 3).

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* President's address.

CASE 4.—Nathaniel M., aged 8 years, admitted to Jefferson Hospital, Jan. 8, 1919. Diagnosis: Cartridge blank in left bronchus. December 24, patient aspirated empty cap of a 22 caliber bullet. Location of foreign body: at orifice of left upper lobe bronchus. Foreign body removed by Dr. Chevalier Jackson, January 11. Time: eight minutes, thirty-one seconds. Recovery (Fig. 4).

It seems only plausible to believe that if one man can see more than seven hundred foreign body cases, and this has been the experience of Dr. Jackson, that very many foreign body cases terminate in death without a correct diagnosis being made, and without any attempt at removal of the foreign body by endoscopy.

The period of latency of symptoms which follows the primary violent dyspnea and choking attack, and later the gradual onset and chronic character of the symptoms suggest to the clinician some acute or chronic pulmonary disease, consequently one not accustomed to the study of these cases might fail to suspect the presence of a foreign body in the lung.

When one reads the histories of these children, their being referred from one competent physician to another, one is struck by the fact that in spite of the history given by the parents of having swallowed or inhaled a foreign object, little attention is paid to this fact, and very often a roentgenogram of the chest is not taken. One of the important lessons I have learned in the last two years from the study of these cases is to suspect a foreign body in the lung, if the following conditions are present: localized lung symptoms that persist in spite of treatment, no tubercle bacilli in the sputum, a leukocytosis for which there seems no definite reason, and a gradual failure in health and weight.

Foreign bodies in the lungs have been regarded practically as curiosities. So far as I can remember only one paper has been read before this society on this subject, and that some years ago. In it, if I remember correctly, three cases were reported, and in the discussion I reported two or three cases that I had found accidentally.

The symptoms of a foreign body in the air passages vary largely, depending on the nature of the foreign body. The peanut kernel immediately sets up a violent dyspnea and an accumulation of purulent secretion in the lungs due to the severe laryngitis, tracheitis, and bronchitis. On the other hand, the inhalation of a metallic foreign body, such as a tack, produces for a very short time violent dyspnea and choking after which there is a period of quiescence in which the patient is entirely free from symptoms for weeks or even months.

The period of quiescence is followed by gradually failing health and strength, cough, moderate fever and a clinical picture that suggests incipient tuberculosis. Undoubtedly foreign body cases have often been so diagnosed.

Metal objects, if they block a bronchus completely, cause a retention of the secretions and may quickly form an abscess. If, however, they occlude the bronchus only partially and the secretions can be coughed up, they may remain for a very long time and do comparatively little damage. The swelling of the mucous membrane, which is always associated, is a factor which ultimately assists greatly in the blocking up of the secretions.

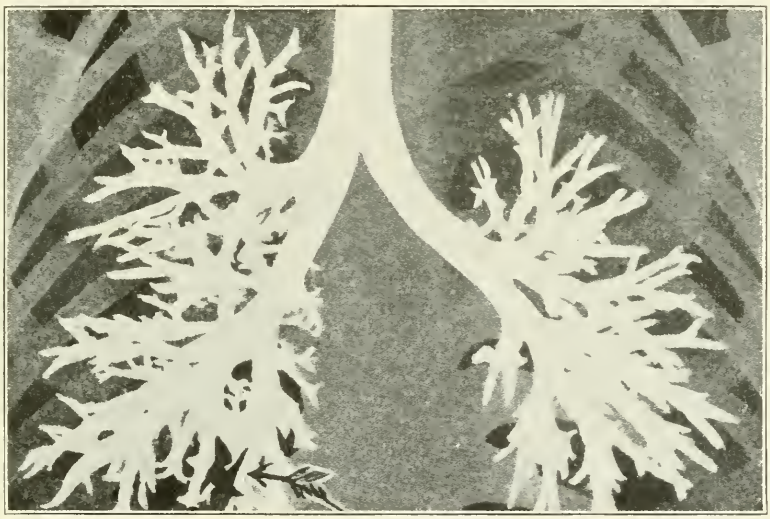

Fig. 1.—Carpet tack in lower lobe of right lung.

Foreign bodies in children are associated with a large amount of secretion. This excess of secretion is probably due both to the fact that the child's lung is more susceptible to the presence of a foreign body than the adult's is, and the fact that the child cannot, like the adult, remove the secretion by coughing. Statistics tend to show that about 66 per cent. of the cases of foreign bodies in the air passages occur in children, due, in my opinion, to the fact that children are very apt to put any and all objects into the mouth and exercise less care in removing them from the mouth than does the adult.

If in the history of the case there is no such history of dyspnea and choking, and one is sure that these symptoms were never present, it is a strong argument against the inhalation of a foreign body.

Food substances, such as bread, lean or fat meat, or those that are apt to be more or less quickly dissolved are usually coughed up and, as a rule, produce no dangerous symptoms. Fine particles of dust are ordinarily coughed up, but large particles, such as coal dust, often become encysted, producing anthracosis. The larger bodies may and probably do produce many of the pulmonary abscesses that are non-tuberculous.

In all foreign body cases a physical examination of the child's entire body, especially of the chest, is most important. Some foreign bodies do not cast a shadow on a photo-sensitized plate, and the study of a large number of such cases ought to be of value. The lung symptoms are nearly always most marked in the lung containing the foreign body. The study of the physical signs, both before and after bronchoscopy, is of importance. The large amount of secretions sometimes removed by the bronchoscopy may cause the signs of consolidation in a lung to disappear. This accumulation of secretions is apt to occur in the lower lobe of the lung, and from a study of a number of such cases it is evident that the secretions gradually accumulate in this lower lobe by gravity, this accumulation taking place in spite of efforts to remove it by coughing.

If a foreign body is present in the esophagus, food may be swallowed easily and regurgitated, or swallowed with difficulty, as is the case in stricture of the esophagus. This is probably due to an associated spasm of the esophagus in the majority of cases, or less often to the change in the position of the body in the esophagus. Sometimes it is impossible to decide on the location of the foreign body in the esophagus from the sensations of the patient. Foreign bodies in the larynx usually produce hoarseness, with a croupy cough and evidences of severe dyspnea. This signifies either glottic or subglottic lodgment.

The asthmatoid wheeze which may be heard in some of the foreign body cases, is, when present, a symptom of considerable importance, especially in those cases where the foreign body does not show a shadow on the plate. It is heard during or at the end of expiration, by placing the ear or stethoscope in front of the mouth of the patient.

It is ill advised to urge a patient to cough with the hope of expelling the object inhaled. The possibility of his coughing it up is very slight, and the probability of his doing injury by forcing the sharp point of the object inhaled, such as a tack, into the laryngeal mucosa is very great.

A larger number of foreign bodies enter the right than the left bronchus, from 62 to 75 per cent. according to different observers. Consequently more foreign bodies lodge in the right lung than in the left, and they rarely lodge in the middle lobe bronchus. There are good anatomical reasons for this, as the diagram from Dr. Jackson's book clearly shows (Fig. 5):

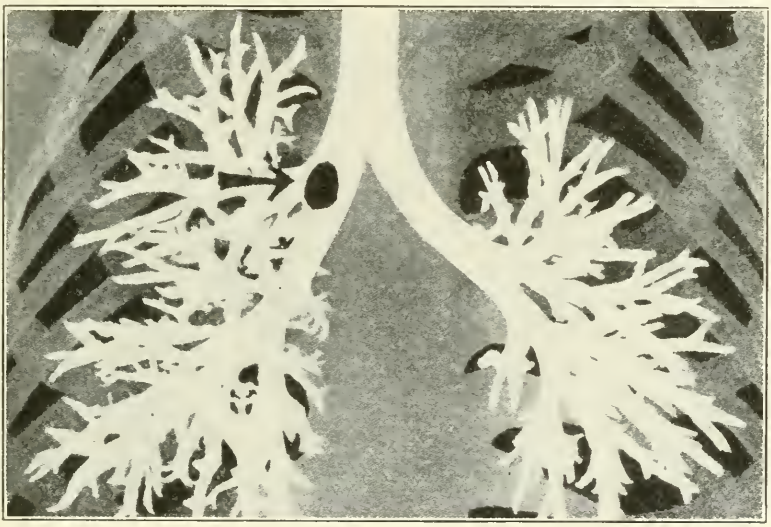

Fig. 2.—Peanut kernel in right bronchus.

The right bronchus is larger than the left, and the angle of deviation is less acute; and the carina, i. e., the top of the bifurcation, is to the left of the middle line of the trachea.

Heavy objects, owing to their weight in comparison to their small size, are rarely coughed up. In fact, very light substances, such as cork, are rarely spontaneously removed by coughing, and the consensus of opinion today is to remove the foreign body by endoscopy.

The physical signs of foreign bodies that have localized themselves in the lung depend on the amount and extent of the lesions. They

may vary according to the composition, form, shape and size of the foreign body, as to whether it blocks the bronchus slightly, largely or completely, and also, to a certain extent, on the amount and character of the secretions. There may be a simple local bronchitis, slight or severe local congestion, slight or much retained purulent secretions, bronchiectasis, or pulmonary abscess. When the foreign body has been present for a long period of time, there is usually a moderate degree of bronchitis in the opposite lung, probably due to the retained secretions gaining access to the other lung, but with the exception of the peanut and maize cases, the local evidences around the foreign body are so well marked that they are usually easily recognized, especially in those cases where the foreign body has been present long enough to produce distinct local pathology. In a case where a foreign body has been present in the lung for a long period of time there is usually considerable cough with offensive expectoration.

If a cavity forms, it may be either a bronchiectatic or an abscess cavity, and there is an absence of tubercle bacilli in the sputum. In a recent case the expectoration is less offensive and more of a mucopurulent character, and occasionally streaked with blood.

Dr. Jackson speaks often of "drowned" lung in his foreign body cases. In a number of cases it is noticed that a great deal of opacity in the tissues surrounding the foreign body disappears after bronchoscopy. This is evidently due to the copious outpouring of secretions after the bronchoscopy. In some of the cases where the foreign body has been present for a number of years, some evidences of damage to the lung may still be visible in the roentgenogram months after the object has been removed, although at this period all physical signs have disappeared clinically. A "drowned" lung, according to Dr. Jackson, is caused by the drainage becoming blocked by the foreign body. The passages of the lungs become filled with secretions, which collect easily, and these consequently become purulent. If this area of drowned lung is not relieved by the removal of the foreign body from the bronchus, we have finally an abscess cavity.

Foreign bodies are occasionally coughed up. It is more likely to be coughed up if the object is in the larynx than if it is in the bronchi. The possibilities of a foreign body being coughed up are very slight, however. The nature of the foreign body has something to do with it. Pins and sharp bodies are rarely if ever expelled by coughing, as the

effort of coughing causes the sharp point to become more tightly fixed. Round, smooth objects usually become firmly lodged, the air below the object is absorbed and coughing consequently can hardly ever be strong enough to disturb the foreign body, as little air is below it. Heavy bodies, as stated before, are seldom coughed up.

According to the experience of Dr. Jackson, pins are the most common foreign bodies in the bronchi, next in frequency are different kinds of hardware, then vegetable substances.

If there is any reason to suspect a foreign body, a bronchoscopy should be performed. It is important to appreciate that a foreign

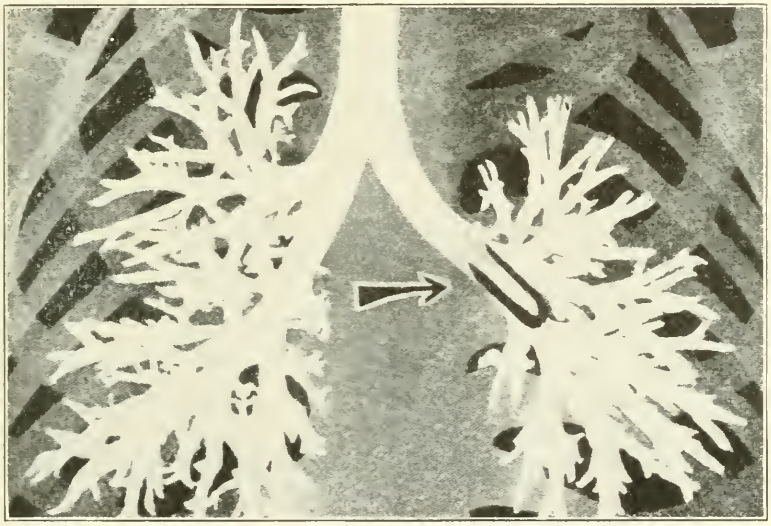

Fig. 3.—Staple in left bronchus.

body does not necessarily produce any physical signs or symptoms, and, on the other hand, it is important to remember that certain lesions in the air passages, as a false membrane, may closely simulate the physical signs and symptoms of a foreign body.

In a case of suspected bronchiectasis, a bronchoscopy may assist in clearing up the diagnosis, especially if the symptoms are limited to one base and are suggestive of tuberculosis, but no tubercle bacilli are found.

There are no contraindications to bronchoscopy, except, perhaps, extreme weakness of the patient, when time should be given the individual to rally partially. Pulmonary abscess, pneumonia, pulmonary gangrene or status lymphaticus, are not contraindications for bronchoscopy.

Bronchoscopy should be performed as soon as possible after the entrance of the foreign body. The longer the time that elapses, the more difficult is the removal owing to the swelling and edema of the bronchial mucosa, later the formation of granulation, and also the fact that the natural tendency of the foreign body is to sink deeper and deeper into the lung. The child's health and strength gradually become impaired, which, of course, tends to make convalescence more tedious.

I have never seen Dr. Jackson give an anesthetic to children, although we have occasionally given a small dose of morphin with atropin to older children before the bronchoscopy.

The necessity of making a roentgenogram in every case in which a history of having swallowed or inhaled a foreign body is given cannot be emphasized too strongly. If a suspicion exists that a foreign body may be present, a roentgenogram should be made, or if an unexplained leukocytosis is present, connected with local physical signs in the lungs that do not clear up under treatment, and if there are no tubercle bacilli in the sputum, the necessity for taking a roentgenogram is evident. It is difficult to explain why so many patients with definite local lesions in one lung that are nontuberculous and that do not disappear under treatment, are not given the benefit of roentgen-ray examination. The probable explanation is that the medical profession, as a whole, does not appreciate the frequency of foreign bodies in the lungs, and the history of having swallowed or inhaled a foreign body is disregarded owing to the period of latency which exists between the time of inhalation and the first development of symptoms.

In a roentgenogram one side may be dark and the other side light. The dark side usually contains the foreign body, perhaps occluding a large bronchus, which may produce a well marked emphysema in the unobstructed lung. It is well to remember that calcified glands may show a rounded shadow.

It is important to appreciate that a negative roentgenogram does not absolutely prove that a foreign body is not present. Metal objects

may not show, or the object may not be opaque to the roentgen ray. The physical signs in such a case are often a valuable guide.

The location of a foreign body in the esophagus that shows but slightly, if at all, in the roentgenogram may be facilitated by having the patent swallow a capsule containing bismuth. This capsule may lodge just above the foreign body. It is important to remember that in a perfectly free esophagus the capsule may stop for a moment in its passage through the esophagus.

If the foreign body is in the lung, the bronchoscope, and if in the esophagus, the esophagoscope is used. Both are passed through the mouth. The illumination is at the distal end, and all the work is done by sight; nothing is done in a blind manner.

The extraction of foreign bodies of steel or iron by the magnet has very narrow limitations, practically only such bodies can be removed as are easily removed by bronchoscopic means.

The bronchi do not diminish in size between the branches. The lessening in the lumen occurs at the point of subdivision between the branches. The lumen between the branches is of a uniform size and does not taper, consequently a foreign body usually is checked and stops below where branching begins.

The diagnosis is made from the history of a foreign body being in the mouth, with an attack of violent dyspnea, and choking sensations. Except when substances such as a peanut kernel or maize is inhaled, this is followed by a period of quiescence and freedom from all symptoms that often lasts for weeks or months. The physical signs are largely localized to a comparatively small area in one lung.

As regards prognosis, Dr. Jackson reports 98.1 per cent. successful removals. His mortalty during operation is less than 0.5 per cent. Including all deaths within one month after endoscopy in more than 700 cases, his mortality is 1.9 per cent. Even this low mortality, if studied carefully, will be found to be due almost entirely to the fact that the patients either were almost moribund when the attempt to remove the foreign body was made, or else the object had been present in the lung so long that notwithstanding its removal the patient later succumbed to the lung condition.

In Jackson's twenty-three cases where the foreign body had been present for from two months to twenty-six years, there were two deaths. The recovery from the pulmonary conditions present is, as a

rule, complete, as far as symptoms are concerned. Cough, expectoration and fever disappear, the child gains in weight and strength, and recovery is apparently complete. The later roentgenologic study of the lungs in these cases also shows a remarkable and to the clinician often an unexpected recovery.

In regard to prophylaxis, all children should be prevented as far as possible from putting any object in the mouth, and when old enough they should be taught the danger of putting objects in the mouth. Great care should be taken that no foreign body gains access to the food before it is eaten. Bones, pins and numerous small objects, such

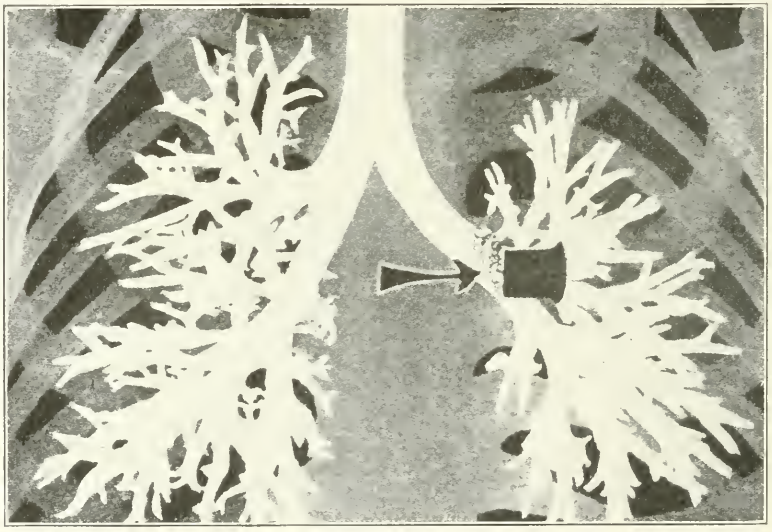

Fig. 4.—Cartridge blank in left bronchus.

as china and glass, are found in foodstuffs, and as a result may be swallowed or inhaled, if not noticed during mastication. It is most important not to leave objects small enough to be placed in the mouth lie where young children can pick them up. A good sized safety pin is not too large to be placed in the mouth of the young child, and possibly swallowed or inhaled.

In children less than 2 years of age a bronchoscopy should not last more than thirty minutes, unless it is urgently required to prolong it. when it may last for one hour. Children more than 2 years of age will

easily stand a bronchoscopy lasting one hour. This is done without either general or local anesthesia, although occasionally a small preliminary dose of morphin and atropin is given. Many of Dr. Jackson's operations for removal of foreign bodies are done in two or three minutes.

As regards treatment, with an early roentgen-ray examination and a bronchoscopy, the treatment is most satisfactory. If a foreign body is inhaled, the patient should be kept as quiet as possible, and as much in the recumbent position as possible, until after the larynx has been examined by the mirror and a roentgenogram has been taken. The idea in keeping the patient recumbent is to keep the foreign body from gravitating lower into the lung. It is important to quiet the patient as much as possible, and he should never be urged to cough with the idea of possibly coughing up the foreign body. It is never wise to pass a bougie into the esophagus for the location of a foreign body or where disease is present. The passage of the bougie practically gives us very little knowledge, and the passage of the esophagoscope, which is certainly no more difficult, gives us valuable knowledge. As a second "don't" to the passage of the bougie might be mentioned the fact that the child should never be held up by the heels and slapped on the back, as there is danger of the foreign body becoming detached, and asphyxiation may result from the object becoming caught in the glottis. Of course, it goes without saying that in all cases of foreign body a roentgenogram should be taken, and taken as early as possible.

All children should be kept in bed and, if possible, under a competent nurse for several days after the foreign body is removed.

As a rule, there is little or no fever after the bronchoscopy, although at times the temperature rises to 100 F. Usually, even in these cases, it falls to normal in a day or two. This was a matter of surprise to me in the first few bronchoscopies that I saw Dr. Chevalier Jackson do, as I expected fever and cough to follow the passage of the bronchoscope, and the manipulation with the instruments necessary to remove the foreign body. The instruments used are, of course, all sterile, and the sensitiveness of the deeper bronchi is very slight as compared with the larynx, hence the child stands the necessary intrusion of the deeper structures well and there can be no infection, as no local or constitutional symptoms follow the operation. If pneumonia is present before the bronchoscopy there may be a severe reaction, although in none of such cases that I have seen has it been fatal.

If before the bronchoscopy, a bronchitis, tracheitis or laryngitis with fever is present, these symptoms are usually aggravated temporarily by the bronchoscopy. The reaction was most marked in the peanut cases. If the epithelium is injured either by the foreign body or by the necessary manipulation, the reaction may be severe following the passage of the bronchoscope. A moderate degree of hoarseness is often seen in children, which usually lasts only two or three days. What Dr. Jackson speaks of as a "drowning of the patient by his own secretions" has been noted in a number of cases in children, especially in the peanut cases. This is usually relieved by the passage of the bronchoscope, and the "sponge pumping" process.

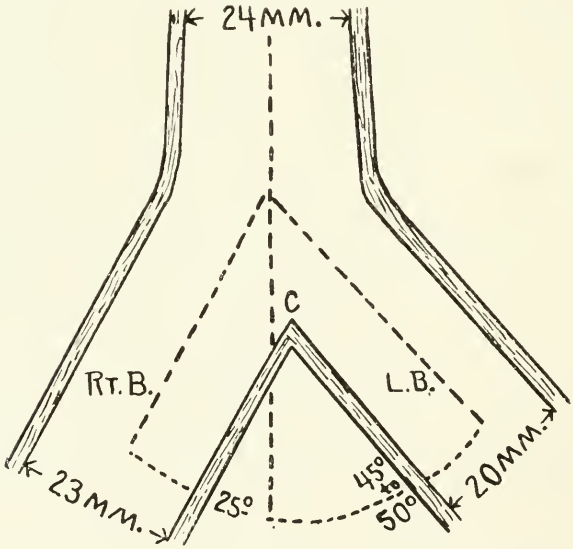

Fig. 5.—Diagram showing three anatomic reasons for the greater frequency of right sided lodgment of foreign bodies in the bronchi. The right bronchus (Rt. B.) is almost as wide (23 mm.) as the trachea (24 mm.), and it deviates from the long axis of the trachea much less than the left. The carina (C) is to the left of this axis. (After Sir St. Clair Thomson.)

Subglottic edema may be present before the bronchoscopy, due in some cases to the presence of a foreign body in the subglottic region, or in the trachea, or it may be caused by the cough or respirations moving the foreign body backward and forward. Jackson believes that distal illumination and the use of small tubes lessens the risk of the

production of this edema of the glottis. If the symptoms are due to subglottic edema a low tracheotomy gives great relief, as I have seen in a number of cases. The tube can usually be removed in a comparatively few days.

CONCLUSIONS

1. It seems reasonable to believe that foreign bodies in the air and food passages in children are much more common than was formerly supposed. Statistics tend to show that about 66 per cent. of these cases of foreign bodies in the air passages occur in children.

2. The period of latency of symptoms which follows the violent dyspnea and choking attack, and later the gradual onset, and chronic character of the symptoms, might lead one to fail to suspect the presence of a foreign body.

3. Foreign bodies are certainly often overlooked, as a study of the histories of many cases clearly shows.

4. The symptoms vary greatly. The peanut kernel immediately sets up a severe laryngitis, tracheitis and bronchitis. In the peanut cases, the older child may survive the acute symptoms, but almost surely will develop pneumonia.

5. Metal objects may remain in the lung for a very long while and do comparatively very little damage.

6. There are some foreign bodies that do not cast a shadow on the plate.

7. The location of a foreign body in the esophagus that does not cast a shadow on the plate may often be diagnosed by allowing the patient to swallow a bismuth filled capsule. The roentgen ray shows the location of the bismuth capsule held in position in the esophagus by the foreign body.

8. Do not urge the patient to cough with the hope of expelling the foreign body inhaled in the lungs.

9. Foreign bodies are very rarely coughed up.

10. The physical signs and symptoms vary according to the composition, form, shape and size of the foreign body.

11. One should suspect a foreign body if the following conditions are present: an unexplained leukocytosis, localized symptoms in one lung that do not clear up under treatment, no tubercle bacilli in the sputum, and a gradual failure in weight and strength.

12. There are no contraindications to bronchoscopy except, perhaps, extreme weakness in the patient, when time should be given the individual to rally partially.

13. Bronchoscopy should be performed as soon as possible after the entrance of the foreign body.

14. Children do not require the administration of an anesthetic for the performing of a bronchoscopy.

15. The necessity of taking a roentgenogram in every case with a history of swallowing or inhaling a foreign body, cannot be emphasized too strongly.

16. The asthmatoid wheeze, when present, is a sign of considerable importance.

A STUDY OF THE LACTOSE, FAT AND PROTEIN CONTENT OF WOMEN'S MILK*

W. DENIS, PH.D., AND FRITZ B. TALBOT, M.D.
(With the assistance of A. S. MINOT)

BOSTON

While the limits and variations in the fat and protein content of human milk are well established, considerable uncertainty still exists regarding the question of the amount of lactose in this fluid,[1] a state of affairs largely due to the fact that while reliable and relatively simple methods for the determinnation of fat and protein have been in use for many years, the question of the technic of sugar determinations in milk is still unsettled. On account of the relatively large quantities of material required when lactose in milk is to be determined either by the polariscope or by one of the copper reduction methods, it is a more or less general custom, when the amount of material is limited, to calculate lactose by difference after having first determined the total solids, ash, protein and fat, a procedure which throws all the errors of the analysis on the sugar.

Last year Folin and Denis[2] described two methods for the determination of lactose in milk, one a colorimetric procedure based on the reduction of picric acid when treated with sodium carbonate and lactose at the temperature of boiling water, the other a titration method based on the reduction of copper in phosphate solution as described by Folin and McEllroy.[3] These methods have the advantage over most of the procedures now in use, first that by means of them it is possible to obtain results of a considerable degree of accuracy in a relatively short time, and second that only 1 or 2 c.c. of human milk is needed for duplicate determinations.

During the past year we have been engaged by the help of the titration method in the collection of data regarding the lactose content of

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* From the Biochemical Laboratories and the Department of Pediatrics of the Harvard Medical Schools and of the Massachusetts General Hospital.

1. Holt, Courtney and Fales: Am. J. Dis. Child. **10**:229 (Oct.) 1915.
2. Folin and Denis: J. Biol. Chem. **33**:521 (March) 1918.
3. Folin and McEllroy: loc. cit., 513.

human milk, and for the sake of completeness have included determinations of the fat and protein.

When the samples of milk were sufficiently large, fat was determined by means of the Babcock method; when the amount of material was limited the analyses were made by Bloor's [4] nephelometric method which requires only 1 c.c. of milk for duplicate determinations.

Protein was calculated from the total nitrogen values obtained by means of the Kjeldahl-Gunning method, the factor 6.25 being employed.

The experimental work of these investigations was divided into four lines and consisted, first, of a study of the composition of milk at different stages of lactation, the results of which are presented in Table 1; second, of a study of the composition of milk at the beginning and at the end of a single nursing, the results of which are presented in Table 2; third, of a study of the composition of milk taken practically simultaneously from the right and left breasts, the results of which are presented in Table 3, and fourth, of a study of the variations in composition of milk taken at three-hour periods during the course of a single day.

The samples of milk were obtained from the Wet-Nurse Directory, the Boston Eye and Ear Infirmary, the Children's Medical Out-Patient Department of the Massachusetts General Hospital, and from private practice. They were either drawn or expressed from a single breast at a time. The following technic was found to be most satisfactory: draw or express one ounce of milk before the infant is allowed to nurse, then allow the baby to nurse the usual length of time, but never longer than twenty minutes; finally draw or express one ounce of milk after the infant has finished. In this manner samples of the "fore milk" and a sample of the "strippings" are obtained. The figures which are reported later show how essential this is and how faulty conclusions may be drawn from the analysis of milk obtained in an improper manner.

Table 1 gives figures averaged from two samples of milk, one taken before nursing and the other immediately after nursing, except in a few instances, noted in the remarks.

It was found that the amount of lactose was low in colostrum, as has already been described by other investigators. In one instance in which the mother had pneumonia, it was necessary to pump her breasts for six days. The total amount of milk diminished until only one

4. Bloor: J. Am. Chem. Soc. 36:1391, 1914.

TABLE 1.—Analyses of Breast Milk at Different Stages of Lactation

No.	Lactose per Cent.	Fat per Cent.	Protein per Cent.	Age of Child	Age of Mother Years	Number of Pregnancies	Remarks
172	3.2	....	....	2 days	..	.....	Colostrum
173	7.5	0.8	....	4 days	..	.....	Has never nursed
176	6.7	3.8	2.1	5 days	..	.....	Milk deep yellow color. Changing from colostrum
75	5.8	....	....	5 days	23	First	Mother has pneumonia. Baby not nursing
76	5.1	1.5	3.7	6 days	..	.....	Same as 75
77	4.85	1.81	2.01	11 days	..	.....	Same as 76
22–23	6.6	1.5	1.57	14 days			
151–2	7.05	3.3	1.30	17 days	26		
147	7.46	2.43	1.59	17 days	24	Third	
149	6.97	3.16	1.83	17 days	..	.....	Seven months premature
72	7.18	1.5	1.97	18 days	20	First	
90	6.45	1.66	1.96	21 days	21	First	Sample taken before nursing
16–17	6.40	5.8	1.18	21 days	16	Second	Baby gaining normally
20	6.73	9.9	1.18	21 days	16	Second	Baby gaining normally
36–7	6.8	1.8	1.40	21 days	20	First	
38	6.93	3.8	1.48	21 days	18	First	Fat and protein sample
47	7.10	6.4	1.48	21 days	..	.....	Sample before nursing
56	7.20	9.5	1.44	21 days	..	.....	Sample before nursing
131	7.07	4.20	1.58	25 days	23	First	
129	7.20	4.00	2.25	26 days	25	Second	
154	6.94	1.81	1.50	25 days	20	First	
42	6.88	4.65	....	4 weeks	..	First	Child has eczema of face
142	7.20	2.6	1.27	4 weeks	..	First	Unknown whether before or after nursing or mixed milk
40	6.96	6.0	1.16	4 weeks	16	Second	Baby gaining, satisfied. Digestion good
43	6.68	7.4	1.05	4 weeks	16	Second	Was given 75 grams lactose 3 hours before nursing
45	6.73	6.0	1.35	4 weeks	18	First	Was given 75 grams lactose 3 hours before nursing
58	6.73	9.2	1.42	4 weeks	18	Second	
60	5.49	6.4	2.23	4 weeks	18	First	Fat and protein from a single sample taken before nursing
156	6.80	3.1	1.63	4 weeks	19	First	
162	6.87	2.18	1.35	4 weeks	21	Third	
145	6.90	3.80	1.48	5 weeks	29	Third	
33	7.05	8.8	1.83	5 weeks	..	Second	Mother producing more milk than baby can take and drawing 12-14 ounces
81	7.00	6.3	1.63	5 weeks	18	Second	
83	6.47	7.05	1.80	5 weeks	22	First	Protein from before nursing, sample only
102	8.20	....	1.56	5 weeks	23	Third	
128	7.2	2.2	1.14	5 weeks	25	First	Single sample taken before nursing
91	6.53	6.36	1.46	6 weeks	16	First	
160	6.04	2.25	1.41	6 weeks	35	Third	
88	6.45	4.9	1.74	7 weeks	21	First	
124	7.8	0.8	1.43	8 weeks	..	.....	Single sample taken before nursing
31	7.43	3.4	1.26	9 weeks	..	Second	
24	6.74	....	....	9 weeks	33	First	
28	6.75	5.0	1.57	9 weeks	38	First	Not quite enough milk for baby
93	6.30	9.9	1.67	9 weeks	22	First	
32	6.94	3.8	1.80	11 weeks	..	Second	Baby weaned because of pneumona. Milk drawn
136	7.70	5.3	1.18	13 weeks	17	First	
79	7.10	1.64	1.37	18 weeks	28	First	Baby has eczema
135	7.30	0.79	1.05	18 weeks	21	First	Single sample before nursing
153	7.20	3.3	1.05	20 weeks	..	Third	Single sample before nursing
108	8.1	3.0	1.18	22 weeks	..	Second	Baby well. Stools regular with soft curd
117	7.80	2.7	1.22	22 weeks	..	.....	Single sample before nursing. Baby has bronchitis and diarrhea
14	6.45	....	....	25 weeks	..	.....	Mother has diabetes; urine contains 6.95% sugar
158	7.20	2.40	1.22	26 weeks	35	Third	
4	6.98	....	....	28 weeks	30	First	
7	7.36	....	....	28 weeks	20	First	
8	6.66	....	....	28 weeks	26	Third	
220	7.6	4.00	1.16	29 weeks	35	Third	Normal mother and infant
12	6.76	....	....	30 weeks	24	First	
116	8.10	2.2	1.00	30 weeks	..	.....	Single sample before nursing. Baby well
104	8.00	....	1.14	32 weeks	23	First	Fat in sample after nursing, 8.4%. Baby had one normal stool a day
110	7.70	....	1.60	24 weeks	..	Third	One normal stool a day
133	7.30	9.3	1.06	39 weeks	..	.....	Normal baby with normal stools

TABLE 1.—Analyses of Breast Milk at Different Stages of Lactation—(*Continued*)

No.	Lactose per Cent.	Fat per Cent.	Protein per Cent.	Age of Child	Age of Mother Years	Number of Pregnancies	Remarks
106	8.35	4.13	1.09	40 weeks	..	Third	Baby 17 pounds. General condition fair; two yellow stools a day
114	8.09	5.33	1.22	40 weeks	..	Sixth	Stools normal
118	7.70	3.9	1.20	43 weeks	..	.....	Baby under weight has bronchitis and green stools
120	7.25	4.25	1.15	49 weeks	..	.....	From 2 to 3 well digested soft yellow stools a day

TABLE 2.—Analysis of Milk at the Beginning and at the End of a Single Nursing

No.	Before			After			Age of Child	Age of Mother, Years	Number of Pregnancies
	Lactose per Cent.	Fat, per Cent.	Protein, per Cent.	Lactose, per Cent.	Fat, per Cent.	Protein, per Cent.			
16, 17	6.09	5.3	1.18	6.72	6.4	1.18	3 weeks	18	Second
20, 21	6.82	8.9	1.18	6.64	10.9	1.22	3 weeks	18	Second
22, 23	6.62	1.5	1.57	6.77	5.2	1.57	2 weeks	18	First
24, 25	7.23	....	....	6.25	....	....	9 weeks	33	First
28, 29	6.86	5.0	1.57	6.64	31.4	1.57	9 weeks	38	First
30a, 30	7.2	0.58	1.40	6.00	7.40				
34, 35	5.45	6.70	1.84	6.64	7.10	1.57	17 days	18	First
36, 37	6.97	1.8	1.40	6.64	....	....	3 weeks	20	First
38, 39	7.04	3.8	1.48	6.82	....	....	3 weeks	18	First
40, 41	6.79	7.1	1.13	7.13	4.9	1.18	4 weeks	16	Second
43, 44	6.94	7.4	1.05	6.43	7.4	1.05	4 weeks	16	Second
45, 46	7.03	4.0	1.65	6.98	8.0	....	4 weeks	18	First
47, 48	7.10	6.4	1.48	4.88	16.0	....	3 weeks	18	First
49a, b	7.18	2.4	1.44	7.62	8.0	1.52	27 weeks		
56, 57	7.20	9.5	1.44	6.57	....	....	3 weeks	18	First
58, 59	6.82	7.4	1.53	6.64	11.0	1.31	4 weeks	18	Second
60, 61	5.16	6.4	2.23	5.82	....	....	4 weeks	18	First
62, 63	5.82	5.6	1.97	5.96	5.2	2.2			
67, 68	6.4	2.4	1.83	6.47	3.8	1.92			
70, 71	7.58	2.5	1.66	7.40	4.4	1.57	26 weeks	20	Second
72, 73	7.28	1.6	1.97	7.08	1.4	1.98	18 days	20	Second
79, 80	7.50	2.06	1.44	6.7	1.22	1.31	18 weeks	28	First
81, 82	7.0	5.60	1.57	7.0	7.10	1.70	5 weeks	18	Second
83, 84	6.55	5.6	1.80	6.4	9.1	....	5 weeks	22	First
88, 89	6.65	3.2	1.79	6.26	6.6	1.70	7 weeks	21	First
91, 92	6.90	4.52	1.35	6.16	8.00	1.57	6 weeks	16	First
93, 94	6.53	6.48	1.70	6.06	13.3	1.63	9 weeks	22	First
95, 96	6.66	2.48	1.26	6.30	5.40	1.31			
97, 98	6.87	2.76	0.96	6.62	....	0.96			
99, 100	8.80	1.5	1.22	8.40	2.9	1.18			
102, 103	8.20	....	1.43	8.10	....	1.69	5 weeks	23	Third
104, 105	8.00	....	1.14	7.90	8.4	1.10	32 weeks	23	First
106, 107	8.50	3.50	1.09	8.20	4.76	0.89	40 weeks	..	Third
108, 109	8.00	3.20	1.18	8.20	2.70	....	22 weeks	..	Second
110, 111	7.80	....	1.00	7.50	1.09	....	34 weeks	..	Third
112, 113	8.00	4.20	1.35	7.70	7.24	1.44			
114, 115	8.09	0.66	....	8.09	10.0	1.22	40 weeks	..	Sixth
118, 119	7.70	4.20	1.09	7.70	3.6	1.31	43 weeks		
120, 121	7.00	1.5	1.22	7.50	8.0	1.09	49 weeks		
129, 130	7.20	2.9	2.30	7.10	5.1	2.20	26 days	25	Second
131, 132	7.10	4.20	1.66	7.00	4.10	1.50	25 days	23	First
133, 134	7.30	5.50	1.06	7.30	13.1	1.05	39 weeks		
138, 139	7.30	2.9	1.13	6.60	5.5	1.30			
145, 146	7.00	3.0	1.44	6.80	4.5	1.53	10 weeks	29	Third
147, 148	7.72	1.66	1.61	7.20	3.20	1.57	3 weeks	24	Third
149, 150	7.08	2.64	1.83	6.86	3.68	1.89	3 weeks	..	First
151, 152	7.20	0.10	0.87	6.90	3.30	1.30	2 weeks	26	Third
154, 155	7.08	1.60	1.40	6.80	2.02	1.40	25 days	20	First
156, 157	6.80	2.60	1.57	6.80	3.60	1.70	4 weeks	19	First
158, 159	7.20	2.00	1.22	7.20	2.80	1.22	26 weeks	35	Third
160, 161	6.30	1.70	1.61	5.78	4.00	1.80	2 weeks	24	First
162, 163	6.54	1.90	1.40	7.20	2.40	1.31	4 weeks	21	Third
180, 181	7.50	8.40	1.60	7.20	8.40	1.70			
182, 183	6.90	1.70	1.60	6.70	4.60	1.90			
184, 185	7.50	1.39	1.35	....	3.68	1.28			
222, 223	7.50	....	1.35	7.50	....	1.35			

ounce could be obtained on the last day. When the baby was five days old the milk contained 5.8 per cent. sugar; when six days old, the milk contained 5.1 per cent. sugar, and when eleven days old, the milk contained 4.55 per cent. sugar (numbers 75, 76, 77). This sequence of events was probably due to the drying up of the milk rather than to the pneumonia, and is suggestive of what may be expected at the time of weaning. Further investigations are necessary to settle this point. The average amount of lactose in sixty samples was 7.19 per cent. There was a general tendency for the concentration of lactose to increase throughout the period of lactation. During the first ten weeks, two thirds of the samples of milk contained less than 7 per cent. lactose. After the tenth week there were four times as many samples which contained between 7 and 8 per cent. as contained less than 7 per cent., and many others which contained over 8 per cent. of lactose. The weakest milk after the colostrum period contained 5.49 per cent. of lactose and the richest 8.35 per cent.

The percentage of fat in the milk of different women varied within wide limits, the lowest mixed milk being 1.5 per cent., and the highest 9.9 per cent. The average amount of fat in the mixed samples was 3 per cent., which is the same as that found in 119 Russian women by Skvortzov,[5] and lower than the 4.5 per cent. found by Engle[6] in ten German wet-nurses. If our wet-nurses only are taken, the average of thirty-five samples is 4.18 per cent. of fat. There does not seem to be any relation between the stage of lactation and the amount of fat in the milk. It seems more probable that the percentage of fat depends more on how completely the breasts are drained and how much of the late milk gets into the sample.

The protein is highest, as would be expected, during the colostrum period. There is a general tendency for the amount of protein to diminish as lactation progresses. It is usually over 1.5 per cent. up to the fourth week and after the twelfth week it averages below 1.2 per cent., the lowest being 1 per cent. So far as we were able to determine with incomplete records, there is no relation between the character of the stools and the amount of total protein.

Table 2 shows the variation in the composition of the milk from the beginning to the end of nursing. There is, as a rule, more lactose at the beginning of nursing than at the end; the difference rarely exceeds 1 per cent.

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5. Skvortzov: Russk. Vrach. **11**:1392; Ref. Chem. Abstr. **7**: No. 18, 1913.
6. Engel: Sommerfield's Handb. d. Milchkunde, Wiesbaden, 1909, p. 810.

The percentage of fat, on the other hand, is much greater at the end of the nursing than at the beginning. One example of extreme variation was found in samples 114 and 115 which showed, respectively, 0.66 per cent. fat before nursing, and 10 per cent. after nursing. As a rule, the differences before and after nursing are not so great, and there was usually less than 4 per cent. difference. An occasional sample was very rich both at the beginning and at the end of nursing, as, for example, Nos. 93 and 94, in which the fat was, respectively, 6.48 per cent. before nursing, and 13.3 per cent. after nursing. Occasionally, two samples, one taken before and one after nursing, showed very little change, and in the rare case the milk after nursing showed less fat than before nursing.

TABLE 3.—ANALYSIS OF MILK TAKEN SIMULTANEOUSLY FROM THE RIGHT AND LEFT BREAST

No.	Right Breast			Left Breast			Remarks
	Lactose per Cent.	Fat, per Cent.	Protein, per Cent.	Lactose per Cent.	Fat, per Cent.	Protein, per Cent.	
85, 87	7.50	2.30	1.60	6.65	6.8	1.60	Right breast taken before nursing; left breast entire quantity
65, 66	7.84	0.73	1.05	7.28	0.63	1.05	Both breasts before nursing
122, 123	7.40	12.0	1.43	7.00	3.80	1.84	Entire amount drawn from both breasts separately
126, 127	7.40	0.7	1.07	7.40	1.4	1.17	
143, 144	7.28	3.5	1.79	7.08	2.4	1.70	
166, 164	7.50	1.6	1.26	7.50	1.5	1.22	Samples taken before nursing } Same
167, 165	7.5	5.0	1.44	7.20	10.0	1.44	Samples taken after nursing } woman
168, 170	7.5	0.80	0.80	7.70	0.8	0.80	Samples taken before nursing } Same
169, 171	7.5	2.4	0.90	7.70	1.9	0.80	Samples taken after nursing } woman
174, 175	6.8	3.3	1.40	7.5	1.8	1.40	Whole of milk drawn, but it did not represent completely empty breasts
30b, 30c	7.2	6.58	1.40	7.2	1.9	1.50	Samples before nursing
18, 19	7.6	2.6	1.18	6.8	8.8	1.01	Right breast normal; left had breast abscess and is scarred

The protein showed less difference in percentage than the fat. It rarely varied more than a few tenths between the beginning and end of nursing. Sometimes it was lower after nursing, and sometimes higher.

The above results emphasize the importance of analyses of milk both before and after nursing, and the uselessness of analyses made (as is too frequently the case) on the first ounce of milk drawn.

Table 3 shows the variation in the composition of the milks taken from the right and left breast of the same woman at about the same

TABLE 4.—ANALYSIS OF BREAST MILK TAKEN AT THREE HOUR PERIODS DURING THE COURSE OF A SINGLE DAY

Time	Lactose, per Cent.	Fat, per Cent.	Protein per Cent.	Volume of Milk C.c.	Remarks
Observation 1	....	....	....	...	Mrs. E. L.: Age of mother 22 years; age of infant 4 months; first pregnancy; normal infant; weight 15 pounds 11 ounces
Nov. 27, 1917					
6 a.m.	7.30	4.0	1.18	180	Breakfast: Cornmeal mush, corned beef hash, muffins, milk and cocoa
9 a.m.	7.25	6.4	1.26	180	Lunch, 10:30 a.m.: Cornmeal gruel with milk
12 m.	7.23	12.5	1.31	170	Dinner: Fish, potato, banana salad, ginger bread
3 p.m.	7.12	8.0	1.22	128	Lunch, 3:30 p.m.: Mellin's food and milk
6 p.m.	7.18	5.3	1.22	157	Supper: Scalloped corn, bread and butter, prunes, cocoa
10 p.m.	7.23	3.6	1.18	150	8 p.m.: Cocoa
Observation 2	....	....	....	...	Mrs. B. R. H.: Age of mother 23 years; baby born May 18, 1918; weight 12 pounds 15 ounces; gained 7 ounces in past week
July 11, 1918					
6 a.m.	6.91	4.00	1.35	202	Breakfast: Oatmeal, hash, potatoes, bread, butter and cocoa
9 a.m.	7.08	4.56	1.40	150	10:30 a.m.: Mellin's food witth milk
12 m.	7.08	5.32	1.40	120	Dinner: Hamburg steak, rice, carrots, cottage pudding, cocoa
3 p.m.	7.08	6.02	1.44	130	3:30 p.m.: Cocoa
6. p.m.	7.28	3.62	1.44	120	Cacaroni, bread and butter, stewed peaches, cocoa
9 p.m.	7.28	2.84	1.49	90	8:30 p.m.: Cocoa
Observation 3	....	....	....	...	Mrs. M. N.: Age of mother 20 years; baby born May 15, 1918; weight 10 pounds 9 ounces; gained 6 ounces in past week
July 11, 1918					
6 a.m.	7.08	4.73	1.26	90	Food same as in Observation 2
9 a.m.	6.91	3.92	1.35	60	
12 m.	6.91	3.36	1.31	150	
3 p.m.	7.08	3.72	1.35	50	
6 p.m.	7.08	4.00	1.49	90	
9 p.m.	7.08	2.80	1.23	75	
Observation 4	....	....	....	. .	Mrs. M. H.: Age of mother 20 years; baby born April 1, 1918; weight 10 pounds 12½ ounces; gained 6 ounces in past week
July 11, 1918					
6 a.m.	6.91	2.00	1.22	135	Food same as in Observation 2
12 m.	6.81	3.72	1.18	172	
3 p.m.	6.91	3.08	1.05	75	
6 p.m.	6.91	4.20	1.09	105	
9 p.m.	6.80	2.48	1.05	75	
Observation 5	....	....	....	...	Mrs. S. McG.: Age of mother 23 years; baby born May 2, 1918; weight 8 pounds 12 ounces; gained 4 ounces in past week
July 11, 1918					
6 a.m.	7.08	2.40	1.67	120	Food same as in Observation 2
9 a.m.	7.08	3.00	1.40		
12 m.	7.20	4.00	1.31	90	
3 p.m.	7.08	3.48	1.40	120	
6 p.m.	7.08	3.48	1.26	75	
9 p.m.	7.09	2.40	1.35	84	
Observation 6	....	....	....	...	Mrs. R. M.: Age of mother 23 years; baby born Dec. 1, 1917; weight 15 pounds 6 ounces; gained 5 ounces in past week
July 11, 1918					
6 a.m.	7.18	2.76	1.05	202	Food same as in Observation 2
9 a.m.	7.00	4.56	1.12	158	
12 m.	6.90	4.56	1.00	120	
3 p.m.	7.20	3.80	1.05	90	
6 p.m.	7.20	3.74	1.00	150	
9 p.m.	7.20	2.85	1.18	120	

time. These figures show that although the milks tend to have the same composition from both breasts, they may be quite different as in Cases 122 and 123, and in Cases 18 and 19 where one breast was scarred by a breast abscess.

There was a tendency for the volume of the milk to diminish as the day went on, the smallest amount being obtained at 3 p. m. or later. There was very little variation in the amount of lactose and protein, and no rule can be devised from these cases. There were striking variations in the amount of fat, especially in Case 1, and there was a tendency for its concentration to be greater at noon or in the afternoon than at other times of day. There was no relation, however, between the percentage of fat found and the amount of milk drawn from the breast.

SUMMARY

1. Newer chemical methods make it possible to quantitate accurately the fat, lactose and protein from small samples of milk.

2. There is a rapid increase of lactose during the first few days when colostrum changes into milk, and a further increase as lactation progresses. The reverse is true of protein which after the first rapid decrease during the change from colostrum into milk tends to further decrease during the course of lactation. After the colostrum period there does not seem to be any relation between the stage of lactation and the amount of fat in the milk.

3. There is usually a higher percentage of lactose at the beginning of a single nursing than at the end. Although this difference may be one or more per cent., it is usually less. It is almost the rule for the percentage of fat to be much higher at the end of nursing than at the beginning. There is very little, if any, difference in the protein.

4. The milks taken simultaneously from both breasts of the same woman tend to have the same composition, but often vary in respect to the percentage of fat.

5. Toward the middle or later afternoon the volume of milk in a woman tends to diminish. The percentage of fat is as a rule higher at mid-day or mid-afternoon than at other times of day.

DISCUSSION

DR. HOLT: I am much interested in the figures Dr. Talbot has given for sugar, which are about what we have found in our laboratory, namely, that sugar in breast milk is quite often 8 per cent. and may be above this. Many disturbances of digestion are attributed to fat which are really due to sugar.

A baby may be gaining eight or ten ounces a week and yet have frequent thin, sour, green stools, and the evidence is that this diarrhea is due to volatile fatty acids derived from sugar. If an examination of the milk is made it will often be found to contain 4 or 5 per cent. fat and as the stools contain an abnormal amount of fat an erroneous inference is drawn that it is the fat that is responsible for the condition. The reason the stools contain an excess of fat is because the fermentation of sugar causes a diarrhea which interferes with fat digestion and absorption and consequently more fat than is usual appears in the stools. These children may continue to gain rapidly in weight yet the condition of the stools seems serious. They improve if taken temporarily off the breast and put on a milk formula less in sugar, or if partly nursed and partly fed on such a formula. We must get away from the idea that breast milk always contains 7 per cent. of sugar or less, and we must accept the fact that disturbances of digestion in nursing infants may be due to sugar. If the child does not vomit we may usually put the intestinal disturbance down as due to sugar rather than to fat. The fatty acids (chiefly lactic and acetic) produced as the result of intestinal fermentation are often so strong as to give the stools a sour odor and cause excoriation of the buttocks.

DR. SHERMAN: I understood Dr. Talbot to say that the protein content of the breast milk bore no relation to the effect of the high carbohydrates. I surely must have misunderstood this statement, for I firmly believe that the addition of some foreign protein, such as powdered casein, will by changing the ratio of the food elements overcome these conditions due to high carbohydrates, which Dr. Talbot has described.

DR. TALBOT: There is one thing I want to impress on your minds and that is that so far as we could determine all these are normal figures. The composition of the milk had no effect on the character of the stools. Those in which we determined that the percentage of sugar after ten weeks was normal did not have watery stools. Dr. Holt has confirmed what our experiments showed some years ago and I think we are all in accord with what he has described when large percentages of sugar are given. These children getting 8 per cent. sugar were without watery stools, so we considered that this was a normal percentage in later lactation. Some of the mothers were given large amounts of candy and also lactose without increasing the lactose in their milk.

THE PATHOGENESIS OF CERTAIN NUTRITIONAL DISORDERS *

(Abstract)

W. McKIM MARRIOTT, M.D.
ST. LOUIS

Some infants suffering from severe diarrhea lapse into a condition simulating an intoxication. Finkelstein, failing to appreciate the true nature of the condition and believing it to be a food poisoning, has referred to it as "alimentär intoxikation." The whole clinical picture may be explained as the result of loss of water and the resulting anhydremia.

The blood of these patients is invariably concentrated, as may be shown by measurement of the protein content of the serum by the refractometer. The diminished blood volume thus brought about leads to a greatly diminished volume flow of the blood and this, in turn, results in diminished oxidation in the tissues and the accumulation of acid products of suboxidation. From such a concentrated blood very little urine is secreted and, as a result, end products of metabolism including acids are retained in the body. We thus have a double explanation of the acidosis and the accompanying symptoms of air hunger and coma.

Blood concentration results in arteriolar constriction and an accumulation of corpuscles in the rather stagnant capillary blood; this explains the peculiar grayish color of the skin of the infants suffering from this condition.

In a certain number of instances the blood pressure falls. This is readily explained on the basis of greatly diminished blood volume. Considerable experimental evidence has been brought forward to show that concentration of the blood can, of itself, lead to a marked degree of fever.

The loss of water necessary to bring about the concentration of body fluids observed is quite sufficient to account for the great and rapid loss of weight invariably observed at the onset of symptoms.

When it is possible to administer sufficient water to cause a return of the blood concentration to normal the symptoms all abate. It is

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* From the Department of Pediatrics, Washington University and the St. Louis Children's Hospital.

not always possible to administer and have retained sufficient fluid. The most effective method of administering fluid, when but little is retained by the alimentary canal, is by intraperitoneal injections of saline at frequent intervals, and intravenous injections of hypertonic glucose, or glucose-acacia solutions.

The symptoms of advanced anhydremia closely resemble those of "wound shock;" in both there is a diminished blood volume as the essential factor. The diminished blood volume is, however, brought about in an entirely different manner in the two conditions.

Anhydremia results from other causes than diarrhea, but no matter what the cause, the symptoms are the same. We have observed it in the course of pneumonia and of scarlet fever, in septic conditions, and as the result of persistent vomiting from parenteral causes, and in idiots who have refused all fluids, etc. Anhydremia is of much more frequent occurrence in warm weather, on account of the increased loss of water from lungs and skin.

In the condition known as marasmus or athrepsia virtual starvation has occurred as the result of a long period of undernutrition. The food stores of the body have been drawn upon to supply fuel and even the blood protein has been, in part, used up. This leads to a diminished blood volume and this in turn to a diminished volume flow of the blood and the accompanyinig symptoms. As the blood is not concentrated there is no fever and no decrease in the urinary secretion. Many of the symptoms are identical with those of anhydremic intoxication, and it is readily understood why these infants so readily succumb to a slightly increased water loss which still further decreases the blood volume.

The blood volume may be restored temporarily to normal by means of transfusion with blood or with a gum acacia solution. Unless food is administered in sufficient quantities to meet the demands of the body such methods as transfusion can afford no more than very temporary relief. Some food may be given intravenously in the form of glucose. The chief reliance must be in some easily digestible food given by mouth. Mixtures of whole lactic acid milk and commercial corn syrup have been most suitable in our hands.

DISCUSSION

DR. SCHLOSS: Pediatric literature contains many references to protein, sugar and fat but contains very little about water. It seems to me that the problem of water retention is at the present time one of the most important with which we are confronted. One phase of this question has been illustrated by Dr.

Marriott's paper. It has been recognized for a great many years that children suffering from intestinal intoxication are in a state of dehydration. Dr. Marriott's paper is particularly interesting inasmuch as he explains all of the symptoms from which these patients suffer on the basis of circulatory disturbances due to diminished blood volume and diminished blood flow.

I have felt for some time that the acidosis occurring in these cases is due to a diminished secretion of urine due to the loss of water and the concentration of the blood. Dr. Marriott's point in regard to the effect of a diminished blood flow on acidosis is a matter of considerable importance. It is some time since I have given bicarbonate to babies suffering from intoxication. It has been a repeated experience that no matter how pronounced the acidosis one can usually correct it by giving very large amounts of saline and 10 per cent. dextrose solution intravenously. This would seem to bear out Dr. Marriott's observations.

DR. ABT: I would like to speak of the possible impression of Dr. Marriott's paper on the pediatrist. You all know that many attempts have been made to classify intestinal disorders, both by clinicians and bacteriologists, and we know how futile they have been. Now the chemists and pathologists are trying their hand, and I hope they will be successful.

The most valuable etiologic classification we have had is that based on food disturbance. It seems to me that by noting the effects of certain foods in various conditions of health and disease the most important basis for the classification of nutritional disorders would be furnished. Dr. Marriott has not referred to this side of the question, and while he has given us an important contribution to the chemistry and pathology, the important question is how to prevent pathology. I would like to ask him whether he has any suggestion concerning the use of foods in disease prevention.

DR. MARRIOTT: In this paper no attempt has been made to discuss the etiology of diarrhea, the end results only have been considered; they are the same no matter what the cause. The prophylaxis of the conditions described is the prevention of diarrhea, and care that the infant receives sufficient food and sufficient water at all times.

A STUDY OF THE RELATIONSHIP OF CONVULSIONS IN INFANCY AND CHILDHOOD TO EPILEPSY

JOHN LOVETT MORSE, A.M., M.D.
Professor of Pediatrics in the Harvard Medical School
BOSTON

About twenty or twenty-five years ago I was much interested in the discussion which was then going on as to the connection between convulsions in early life and epilepsy. I felt that at that time I had no right to an opinion of my own, but resolved to keep the matter in mind and to see later if I could draw any conclusions from my own experience. I have, therefore, looked up, as far as possible, the condition of the babies and children with convulsions whom I have seen in consultation and in private practice. I have included only those cases in which the convulsions were the primary cause for my attendance and have excluded all of those cases in which there were any evidences of acute or chronic cerebral disease. I have had two objects in mind in this study: first, to determine what proportion of the children, otherwise apparently normal, having convulsions have epilepsy or develop it later, and, second, to find out, if possible, whether there is anything in the history or in the manner of the development of the convulsions to show whether or not they are manifestations of epilepsy, or whether they will be followed by or develop into epilepsy later.

I have been able to obtain satisfactory reports regarding 107 children. The time elapsed has varied between two and twenty years. In order to study these cases better and to avoid confusion, I have divided them into four classes: first, those cases in which the convulsions were associated with evidences of spasmophilia; second, those cases in which the convulsions occurred in the course of whooping cough; third, those cases in which there was a single convulsion or a series of convulsions at the onset of some acute disease or with an attack of acute indigestion; fourth, those cases in which there had been repeated convulsions during a considerable period or in which there had been repeated attacks suggesting petit mal. I shall take up each of these classes separately.

1. *Cases Associated with Evidences of Spasmophilia.*—There were ten cases in this class, all occurring in infants. In one instance the convulsions had lasted only twenty-four hours. In the other cases there had been many attacks during two weeks, three weeks, three weeks, one month, two months, three months, three months, seven months and nine months, respectively. Four of these babies had no more convulsions, and one had only one more. Two continued to have convulsions for a week, and one of them intermittently for a year. In another, the attacks persisted for two years. It was finally decided, however, that these attacks had changed in character, and that the child induced them voluntarily in order to obtain its wishes. They stopped at once, never to return, when a glass of cold water was thrown in the child's face at the beginning of an attack. In another instance, the attacks persisted for two years and then ceased. This child, however, became feebleminded. It is probable, therefore, that in this instance the convulsions were not a manifestation of spasmophilia, but were due to some other cause. This supposition is the more probable, because the first attacks appeared coincidently with an attack of influenza and otitis media. She died of infantile paralysis when 9 years of age. All the other children are now alive and well. The time elapsed since these children were seen has been four, five, seven and one-half, eight, nine and one-half, eleven and one-half, twelve and one-half, twelve and one-half, fourteen and one-half and twenty years, respectively.

It would seem, therefore, that the convulsions which occur in babies with spasmophilia, and which are presumably a manifestation of spasmophilia, are not likely to lead to the development of epilepsy later.

2. *Cases in Which the Convulsions Occurred in the Course of Whooping Cough.*—There were three cases in this class. In one case, a child 6 years of age, there was only one convulsion. She was unconscious, however, for several days. She has been and is now well, three years later. Another, a baby four months of age, had several convulsions in twelve hours. The child has been and is now well, eleven and one-half years later. The third patient, a baby, sixteen months of age, had two convulsions in four days. She developed epilepsy two years later and still has attacks, sixteen and one-half years after the attack of whooping cough. She is normal mentally and physically.

TABLE 1.—Cases in Which There Had Been Repeated Convulsions During a Considerable Period or in Which There Had Been Repeated Attacks Suggesting Petit Mal

A. EPILEPTICS

No.	Age When Seen	Age at Onset	Duration When Seen	Nature of Attacks	Apparent Etiology When Seen	Diagnosis When Seen	Labor	Possible Etiology	Results
1	8 yrs.	5 yrs.	3 yrs.	Petit mal	None	Epilepsy or hysteria	Normal	None	Grand mal, feeble-minded
2	10 yrs.	Infancy and again at 10 yrs.	10 yrs.	General	Indigestion	Reflex convulsions	Normal	None	Normal for 7 yrs., then grand mal
3	3½ yrs.	3½ yrs.	5 wks.	General	None	Reflex convulsions or epilepsy	Forceps	None	Attacks continued, grand mal
4	3¾ yrs.	17 mos.	2 yrs.+	General	Indigestion	"Reflex convulsions" or epilepsy	Normal	None	Normal for 6 yrs., then petit mal
5	5 yrs.	5 mos.	One convulsion; petit mal beginning one month before seen	Petit mal	None	Petit mal	Breech, hard to resuscitate	Injury at birth or first convulsion	Grand mal
6	10 yrs.	1 yr.	9 yrs.	General	None	Epilepsy	Normal	None	Grand mal
7	7½ yrs.	6 mos.	One convulsion; petit mal beginning six months before seen	Petit mal	Injury from first convulsion?	"Petit mal" or reflex convulsions	Normal	Injury from first convulsion	Grand mal, feeble-minded
8	18 mos.	13 mos.	5 mos.	General	Indigestion	"Reflex convulsions" or epilepsy	Normal	None	Grand mal, feeble-minded
9	5 yrs.	2 yrs.	3 yrs.	General	Injury to head from fall	"Epilepsy" or reflex convulsions from indigestion	?	Injury from fall	Grand mal, feeble-minded
10	4 yrs.	3½ yrs.	6 mos.	General	Indigestion	Epilepsy	Normal	None	Petit mal
11	5 yrs.	1½ yrs.	3½ yrs.	General and petit mal	Indigestion	"Reflex convulsions" or epilepsy	Hard	None or hard labor	Petit mal
12	12 yrs.	11 yrs.	1 yr.	General	Injury to head from kick of horse or nervousness	"Reflex convulsions" or epilepsy	?	Injury to head	Grand mal
13	10 mos.	5 mos.	5 mos.	General	Indigestion	"Reflex convulsions" or epilepsy	Hard	Hard labor	Grand mal, feeble-minded
14	5 yrs.	1 yr.	4 yrs.	General	None	Epilepsy	Normal	None	Grand mal
15	2½ yrs.	1½ yrs.	1 yr.	Petit mal	Hard labor	Petit mal	Hard	Hard labor	Grand mal
16	7 yrs.	1½ yrs.	Two convulsions at 18 months; petit mal beginning one year before seen	Petit mal	Hard labor or first convulsion	Petit mal	Hard	Hard labor or first convulsion	Grand mal
17	7½ yrs.	Birth	Two convulsions in first 24 hours, two in next four days; no more until one week before seen; from fright	General	Fright	Eclampsia, not epilepsy	Hard	Hard labor or first convulsion	Grand mal

TABLE 1.—Cases in Which There Had Been Repeated Convulsions During a Considerable Period or in Which There Had Been Repeated Attacks Suggesting Petit Mal—*Continued*

B. NOT EPILEPTICS

No.	Age When Seen	Age at Onset	Duration When Seen	Nature of Attacks	Apparent Etiology When Seen	Diagnosis When Seen	Labor	Time Since Seen	Time Since Last Convulsion	Remarks
18	3 yrs.	2 yrs.	1 yr.	General, 5; fainting attacks also	Indigestion	Reflex convulsions	Normal	4 yrs.	2 yrs.	Two in next 2 years, from indigestion
19	6 mos.	Birth	6 mos.	General, many	Otitis media, indigestion	Reflex convulsions	Forceps	3 yrs.	2 yrs.	Continued for 1 year, until cut last tooth
20	3 yrs.	1½ yrs.	1½ yrs.	General, 3	Indigestion	Reflex convulsions	Normal	3 yrs.	3 yrs.	No more convulsions
21	6 yrs.	2 yrs.	4 yrs.	Petit mal, 7	Indigestion	"Reflex convulsions" or epilepsy	Forceps	3½ yrs.	3½ yrs.	One slight attack soon after
22	8 mos.	6 mos.	2 mos.	General, 2 sets	Indigestion	Reflex convulsions	Forceps	3½ yrs.	3½ yrs.	No more convulsions
23	2 mos.	6 wks.	2 wks.	Several severe and many slight	Indigestion	Reflex convulsions	Cesarian	4 yrs.	4 yrs.	No more convulsions
24	7½ yrs.	7 yrs.	6 mos.	General, 6 in last 3 weeks, slight attacks of stiffness in leg for 6 months	Indigestion	"Epilepsy," tumor or reflex convulsions	Normal	9½ yrs.	4½ yrs.	Gradually diminished in frequency and ceased
25	9 mos.	8 mos.	1 mo.	General, 5	Circumcision, 1; indigestion, 4	Reflex convulsions	Forceps	4½ yrs.	4½ yrs	Stopped after a few days
26	2½ yrs.	1½ yrs.	1 yr.	General, 5	Indigestion and onset of acute disease	Reflex convulsions	Forceps	5½ yrs.	4½ yrs.	One or two in next year from indigestion
27	7 yrs.	6½ yrs.	6 mos.	General, 2	Overfatigue and indigestion	"Reflex convulsions" or epilepsy	Forceps	5½ yrs.	5 yrs.	One or two in next 6 months
28	20 mos.	12 mos.	8 mos.	General, 4	Fright or crying	"Convulsions from nervousness" or epilepsy	Forceps	5½ yrs.	5½ yrs.	Two from pertussis soon after
29	3 yrs.	2½ yrs.	6 mos.	Petit mal	Began with an acute illness	"Functional" or epilepsy	Normal	6½ yrs.	5½ yrs.	Gradually diminished in frequency and ceased
30	3 yrs.	2½ yrs.	6 mos.	Petit mal	Indigestion	"Reflex convulsions" or epilepsy	Normal	5½ yrs.	5½ yrs.	Attacks soon ceased
31	9 mos.	8 mos.	1 mo.	General, 1; then many in 24 hrs. 3 weeks later	Indigestion	Reflex convulsions	Forceps	5½ yrs.	5½ yrs.	No more convulsions
32	4 yrs.	2½ yrs.	1½ yrs.	General, 12 in 18 months	Indigestion	"Reflex convulsions" or epilepsy	Normal	7 yrs.	6 yrs.	Occasionally for 1 year

33	2½ yrs.	1 yr.	1½ yrs.	General, 3	Indigestion	Reflex convulsions	Normal	7 yrs.	7 yrs.	No more convulsions
34	9 mos.	Birth	9 mos.	Petit mal	Adenoids or hard labor	"Reflex convulsions" or epilepsy	Hard	10 yrs.	7 yrs.	Occasional attack until adenoids removed
35	2½ yrs.	2½ yrs.	6 wks.	General, 6	Indigestion and otitis media	Reflex convulsions	Normal	7½ yrs.	7½ yrs.	No more convulsions
36	10 yrs.	9 yrs.	1 yr.	General, several; petit mal, many	None	Epilepsy	Forceps	9 yrs.	8½ yrs.	Attacks soon ceased
37	6 mos.	Birth	6 mos.	General, few; many slight attacks of tremor	Injury to mother during pregnancy	"Epilepsy" or reflex convulsions	Forceps	9 yrs.	9 yrs.	No more attacks
38	22 mos.	2 mos.	20 mos.	General	Indigestion	Reflex convulsions	?	9 yrs.	9 yrs.	No more convulsions
39	3 mos.	2 mos.	1 mo.	General, 3	Indigestion	Reflex convulsions	Normal	9 yrs.	9 yrs.	No more convulsions
40	6 yrs.	3 yrs.	3 yrs.	Petit mal	None	Epilepsy	Normal	9 yrs.	9 yrs.	No more convulsions
41	3½ yrs.	2¾ yrs.	9 mos.	General, 4	Indigestion	Reflex convulsions	Normal	9½ yrs.	9½ yrs.	No more convulsions
42	8 yrs.	1 yr.	7 yrs.	General, few	Indigestion	Reflex convulsions	?	10 yrs.	10 yrs.	No more convulsions
43	27 mos.	5 mos.	22 mos.	General, many	Indigestion	Reflex convulsions	?	11½ yrs.	11½ yrs.	No more convulsions
44	7 mos.	2 mos.	5 mos.	General, many	Indigestion	Reflex convulsions	Forceps	11½ yrs.	11½ yrs.	No more convulsions
45	11 mos.	9 mos.	2 mos.	General, many	None	"Reflex convulsions" or epilepsy	Normal	13½ yrs.	11½ yrs.	Attacks diminished in frequency for 2 years, then ceased
46	21 mos.	18 mos.	3 mos.	General, 2	Bronchitis, indigestion	"Reflex convulsions" or epilepsy	Normal	12 yrs.	12 yrs.	No more convulsions
47	4 mos.	3 mos.	1 mo.	General, many	Indigestion	Reflex convulsions	Normal	13½ yrs.	12 yrs.	One attack 1½ years later
48	2 yrs.	18 mos.	6 mos.	General, 2	Indigestion	"Reflex convulsions" or epilepsy				
49	18 mos.	12 mos.	6 mos.	General, 5	Constipation	Reflex convulsions	Normal	12 yrs.	12 yrs.	No more convulsions
50	6 mos.	Birth	6 mos.	General, many	Indigestion or hard labor	Reflex convulsions or "epilepsy"	Normal	12½ yrs.	12½ yrs.	No more convulsions
51	6 yrs.	18 mos.	4½ yrs.	General, many; petit mal, very many	Constipation	"Reflex convulsions" or epilepsy	Forceps	12½ yrs.	12½ yrs.	No more convulsions
52	16 mos.	15 mos.	3 wks.	General, 2	Indigestion	Reflex convulsions	Normal	14 yrs.	14 yrs.	No more attacks, "cured by dilatation of anus"
53	7¾ yrs.	4 yrs.	3¾ yrs.	Petit mal, frequent	None	Epilepsy	?	14 yrs.	14 yrs.	No more convulsions
54	10 wks.	8 wks.	2 wks.	General, 25 to 100 a day	Indigestion	"Reflex convulsions" or congenital cerebral lesion	Normal Normal	15 yrs. 15½ yrs.	14 yrs. 15½ yrs.	Persisted for 1 year No more convulsions
55	2 mos.	3 wks.	5 wks.	General, 2	Indigestion	Reflex convulsions	?	18 yrs.	18 yrs.	No more convulsions

These cases are too few in number to warrant any definite conclusions. They show, however, that convulsions in whooping cough should be regarded seriously, and that they may lead to the development of epilepsy later. They also seem to show that no conclusions can be drawn at the time from the severity or the duration of the convulsions as to whether epilepsy will develop later or not.

3. *Cases in Which There was a Single Convulsion or a Series of Convulsions at the Onset of Some Acute Disease or With an Attack of Acute Indigestion.*—There were thirty-nine cases in this class. The severity of the convulsions varied materially. In some cases there was only one slight convulsion, while in others the children were in a convulsive state for from two to four days. There were all degrees of severity between these two extremes. Thirty of these patients were infants, six were between 2 and 5 years of age, and three were between 5 and 10 years of age.

Two of these children are now epileptic and feebleminded. One of them was in a convulsive state for thirty-six hours as the result of indigestion. The other one, a premature infant, had a series of slight convulsions when four days old without apparent cause. A third child has petit mal. This child had two sets of convulsions two weeks apart, each lasting less than twelve hours. Both sets were attributed to indigestion, which he undoubtedly had at the time. Another baby that had its convulsions when six months of age continued to have "staring spells" for six months, which were called petit mal by some physicians and reflex from gas by others. This child had a convulsion with the onset of mumps when 2½ years of age, but has had none in the last year. It hardly seems fair to call him well after so short a period. Thirty-five patients have not had any more convulsions and are now apparently well. The time elapsed since the convulsions in these cases has been two, two and one-half, two and one-half, two and one-half, three, three and one-half, four and one-half, four and one-half, five, five and one-half, six, six and onehalf, seven and one-half, eight, nine and one-half, ten, ten and one-half, ten and one-half, eleven, eleven and one-half, eleven and one-half, eleven and one-half, twelve, twelve and one-half, twelve and one-half, thirteen, thirteen and one-half, fourteen, fourteen and one-half, fourteen and one-half, fourteen and one-half, fifteen, eighteen, eighteen and one-half and nineteen years, respectively. It may be said that it is not safe to consider these children well unless at least five years have elapsed since

the convulsions. On this basis twenty-seven can be regarded as absolutely well.

4. *Cases in Which There Had Been Repeated Convulsions During a Considerable Period or in Which There Had Been Repeated Attacks Suggesting Petit Mal.*—There were fifty-five cases in this class. Seventeen of these children are now epileptics, and five of the seventeen are feebleminded. Three have only attacks of petit mal. The others have grand mal. The children who now have petit mal, all had grand mal when they were seen, and the children who had petit mal when they were first seen, now all have grand mal. In one instance grand mal developed after a free interval of seven years, while in another case petit mal developed after a free interval of six years. Thirty-eight patients are now well. Twenty-two patients had no more attacks. The time elapsed in these cases since they were seen has been three, three and one half, four, five and one-half, seven, seven and one-half, nine, nine, nine, nine, nine and one-half, ten, eleven and one-half, eleven and one-half, twelve, twelve, twelve and one-half, twelve and one-half, fourteen, fourteen, fifteen and one-half and eighteen years, respectively. The intervals since the last convulsions in the other sixteen cases are two, two, three and one-half, four and one-half, four and one-half, four and one-half, five, five and one-half, five and one-half, five and one-half, six, seven, eight and one-half, eleven and one-half, twelve and fourteen years, respectively. As one case developed grand mal after an interval of seven years, it may, perhaps, be said that it is not safe to call a child well until seven years have elapsed. If seven years is taken as an arbitrary standard, only twenty-three of these patients can be said to be absolutely safe.

I have endeavored by analyzing these cases to determine if there was anything in the history or character of the convulsions in these cases which would aid in determining in a given case whether epilepsy was likely to result in the future or not. The results of this analysis have, however, been rather unsatisfactory. There was (as is shown by Table 2) a somewhat larger proportion of hard labors or deliveries by forceps in the cases which developed epilepsy than in those which did not.

Table 3 shows the character of the attacks and the relative proportions of mild and severe forms in round numbers in the two classes of cases.

There was, therefore, a somewhat larger proportion of general convulsions in those that did not develop epilepsy, while nearly two-thirds of the children that had attacks classified as "petit mal" developed epilepsy. That is to say, these figures seem to show that epilepsy is more likely to develop in children who have attacks classified as "petit mal" than in those who have general convulsions. This conclusion hardly seems a reasonable one. The conclusion is justified, however, that repeated attacks, which would be classified as petit mal, are as likely to be followed by epilepsy as are repeated attacks of general convulsions. The age of the patients at the onset of the attacks was below two years in 70 per cent. of both classes, while

TABLE 2.—CONDITIONS AT BIRTH

Labor	Epileptics per Cent.	Not Epileptics, per Cent.
Normal	47	53
Hard or forceps	41	32
Cesarean	..	2
Unknown	12	13

TABLE 3.—CHARACTER OF ATTACKS

Character of Attacks	Epileptics per Cent.	Not Epileptics, per Cent.
General convulsions	65	71
Petit mal	30	16
Both	6	13

convulsions at birth were twice as numerous in the cases in which epilepsy did not develop later as in those that did. These figures seem to show that babies who have convulsions at birth are not as likely to develop epilepsy as those who first have them later. This conclusion may or may not be justifiable. It is reasonable to conclude from them, however, that convulsions at birth are not as likely to result in epilepsy as has been commonly supposed.

The apparent etiology in these cases at the time when they were seen, as far as there were enough of a kind to make it worth while to group them, is shown in Table 4.

This table seems to show fairly definitely that when there is no apparent etiology, or when the cause of the attacks is apparently an injury or severe labor, epilepsy is far more likely to develop than when the apparent cause is a disturbance in the digestive tract. That is, the absence of any apparent cause or the history of an injury or of a hard labor, in the absence of any other apparent cause, makes the development of epilepsy later quite probable. If the apparent cause is indigestion, the chances are more than two to one that epilepsy will not develop.

TABLE 4.—APPARENT ETIOLOGY WHEN SEEN

Apparent Etiology	Epileptics per Cent.	Not Epileptics, per Cent.*
Digestive tract	35	82
Injuries or hard labor	23	8
None	30	11

* Several possible causes in a number of cases.

Table 5 shows the duration of the attacks before the patients were seen.

TABLE 5.—DURATION OF ATTACKS WHEN SEEN

Duration When Seen	Epileptics per Cent.	Not Epileptics, per Cent.
Less than 1 year	30	66
From 1 to 5 years	40	31
More than 5 years	30	3

It is very evident from this table that the longer the attacks have persisted, the more likely they are to be epilepsy or to eventuate in epilepsy.

While the analysis of these cases shows little which is of apparent value in determining whether or not epilepsy will develop later in children who have had repeated convulsions or repeated attacks classified as petit mal, it seems from an analysis of the diagnoses which were made at the time the children were first seen as if there was something about the impression which was obtained or about the appearance of the children which was of more importance than any-

thing which is shown in the analysis of the various points which have been considered. Table 6 is interesting in this connection.

TABLE 6.—DIAGNOSES WHEN SEEN

Diagnosis When Seen	Epileptics per Cent.	Not Epileptics, per Cent.
Convulsions from fright	6	..
Reflex convulsions	6	53
Reflex convulsions or congenital cerebral	..	3
"Reflex convulsions" or epilepsy	29	24
"Other conditions" or epilepsy	6	5
Reflex convulsions or epilepsy	6	..
"Epilepsy" or reflex convulsions	12	3
"Epilepsy" or other conditions	..	3
Epilepsy and petit mal	35	8

The condition quoted is the more probable diagnosis. Percentages are given in round numbers.

This table shows many mistakes in diagnosis, which is not surprising. It shows also that epilepsy was considered in 88 per cent. of the children that developed epilepsy and in only 43 per cent. of those that did not develop epilepsy, and that epilepsy was the first choice in 47 per cent. of those that became epileptic and in only 14 per cent. of those that did not. The flat diagnosis of reflex convulsions was made in only 6 per cent. of those who became epileptic, while it was made in 53 per cent. of those who did not develop the disease. On the other hand, the flat diagnosis of epilepsy was made in 35 per cent. of those who developed the disease and in only 8 per cent. of those who did not.

COMMENT AND CONCLUSIONS

The results of this study are most unsatisfactory and very few conclusions can be drawn from it. The following conclusions are, perhaps, justifiable to a certain extent. Convulsions which are a manifestation of spasmophilia are not likely to eventuate in epilepsy. Convulsions which occur in the course of whooping cough must always be regarded seriously, as they are quite likely to be followed by epilepsy later. Single convulsions or a series of convulsions occurring at the onset of an acute disease or with an attack of acute indigestion are less likely to be followed by epilepsy than are repeated convulsions during a considerable period or repeated attacks suggesting petit mal Repeated attacks which would be classified as petit mal, or which suggest it, are just as likely to eventuate in epilepsy as repeated attacks of general convulsions. Nothing can be told from the nature of the

early attacks as to the nature of the attacks when epilepsy develops later. When an injury to the head has directly preceded the onset of the attacks or there is no apparent cause for the attacks, epilepsy is more probable than when there is an apparent cause, such as indigestion, for each attack. The presence of an apparent cause for the attacks does not, however, exclude epilepsy. The longer the attacks have persisted, the more probable is the diagnosis of epilepsy. General impressions, which cannot be explained, have a certain value in diagnosis. Finally, and most positively, there is no way to determine immediately when a baby or child has a convulsion, or has had repeated convulsions or repeated attacks suggesting petit mal, whether it has epilepsy or whether it will develop it later.

DISCUSSION

DR. KOPLIK: In the main I agree with Dr. Morse. As to spasmophilia, and we all see any number of these cases, we understand convulsions occurring in children who are otherwise absolutely normal, whether the convulsions occur in the new-born or whether they occur later on. By spasmophilic babies are meant those that have an increased opening anodal reaction to 3 milliamperes or below. In the vast majority of these cases, epileptic convulsions as we understand them do not occur later in life. You have likely had cases in which you tested the anodal opening and closing and determined that you had a normal baby and the child came later on with epilepsy, but those are a vanishing number; usually if you test these babies in this way you can make up your mind whether or not they are true spasmophiliacs. In speaking of spasmophilia we must rule out a large number of babies who have convulsions and whose development is not normal. If they have convulsions without increase of temperature they later may develop epilepsy and feeble-mindedness. When a mother comes with the question, "Will my baby develop epilepsy after having had a few convulsions?" I would say that if the baby is otherwise normal the probability is the baby will not have epilepsy. Epilepsy is not a development but a condition, and unless there is a history of epilepsy in the family we should be cautious how we make a mother uneasy over the prospect of having her child develop epilepsy. Sometimes epilepsy develops in children later in life who have never had convulsions. I had a case the other day in a spasmophilic child 5 years old who had convulsions for the first time. We must be very cautious how we make a diagnosis of epilepsy, for spasmophilia may occur later on in childhood.

DR. DEBUYS: I would like to ask Dr. Morse if in his study he grouped children who had "holding breath spells" with the group of spasmophilias, and if not, has he had any children of that kind who have developed epilepsy later?

DR GRIFFITH: It has been a source of encouragement to me to hear Dr. Morse speak discouragingly regarding the possibility of making a positive diagnosis of the existence or nonexistence of epilepsy in children who show a convulsive condition early in life. It has seemed to me practically impossible in most cases to determine at all what the final outcome will be regarding the development of epilepsy. As a rule, idiopathic epilepsy does not develop

in the first two years of life, and it is safe to assume that convulsions seen at that time are probably due to other causes. This, of course, is well known to all of us, but I wish that our views could be brought to the attention of those who are not pediatrists. I think that neurologists, seeing as they do the older and more severe cases, are very prone to make a diagnosis of epilepsy earlier than seems quite justifiable.

With regard to the convlusions occurring in the new-born, the prognosis in these seems to me to be usually grave. It is extremely likely that a meningeal hemorrhage has taken place, the result of a lesion occurring at birth; and such cases will develop symptomatic epilepsy. So, too, the occurrence of convulsions during pertussis is to be regarded as a possibly serious matter, inasmuch as these may be the result of a meningeal hemorrhage produced by the violence of the coughing. Repeated convulsions occurring in infancy which can be associated in any way with indigestion are likely to disappear as the child grows older. The point especially which I would make is that in conversation with parents physicians should be extremely cautious in making a diagnosis of idiopathic epilepsy while the patient is still an infant.

DR. HEIMAN: I have been called to see children, some in which labor has been instrumental and some in which it has been normal, who five or six days after birth developed spasms and coma, and convulsions. In such cases there may be a venous apoplexy and not an arterial apoplexy as in adults. Injuries resulting from the attempt to pass through the birth canal may in such cases result in blood oozing from the cortical area and then the question arises as to how much of the blood clot will be absorbed and how much will organize. Spasmophiliacs may have convulsions. If the spasmophilia is treated early we may prevent epilepsy. Where there has been a cerebral birth injury it is a question of the amount of hemorrhage and the possibility of absorption. If we know something about these points we will be in a better position to give a prognosis as to whether epilepsy is likely to occur.

DR. HOLT: A few years ago I went through my own private histories much as Dr. Morse has done. His conclusions are borne out by my experience in that a large number of children whom we expected to develop epilepsy did not do so. I think the general practitioner and even the neurologist is more likely to regard these cases of convulsions as epileptic than is the pediatrist. We see convulsions so often and in such a wide variety of conditions. Only a small minority of these children turn out to be epileptic.

In regard to what Dr. Heiman said, not only does great pressure in large children but in premature infants even a moderate amount of pressure may cause meningeal hemorrhage and convulsions as a consequence of which a child may become feeble-minded and later epileptic. Recurring convulsions with a sharp rise of temperature, even if there is not much indigestion present, are seldom serious; but if children have repeated attacks of convulsions without fever and without assignable cause we must regard them with suspicion when we have ruled out tetany.

In Dr. Morse's figures of whooping cough cases I think there are hardly enough data to warrant his conclusions. In whooping cough there is extreme susceptibility of the nervous system and unless there are hereditary influences predisposing or a meningeal hemorrhage occurs during an attack, I do not think we are likely to see epilepsy develop later.

The difficult question in a case of convulsions is that of prognosis. Every mother wants to know what the outcome will be, whether the convulsions are the beginning of a condition that is going to become permanent or whether it

is merely transient. Not only in this condition but in all the diseases of children we should teach our students more regarding prognosis. The tendency at present is to lay undue emphasis on diagnosis and the student is likely to think that when he has made the diagnosis he has about covered the case. There are few problems in prognosis more difficult than the one Morse has brought before us.

DR. FREEMAN: A point that has not been mentioned by previous speakers is that in children under 2 years of age who have marked evidence of rachitis convulsions occur with less provocation than in others, and there is thus probably less likelihood that epilepsy will occur.

DR. ADAMS: I have no statistics, but a good memory, and my conclusions are about the same as those of Dr. Morse. The most important thing is to prevent casting the stigma of epilepsy on a child—a thing that is done too frequently. I have had three children within the last ten days in whom to decide whether or not they had epilepsy. In two I said I thought they did not have it, as they gave no history that would indicate epilepsy. In the other case I thought the child had epilepsy from his history.

In cases of whooping cough one should be cautious in making a diagnosis of epilepsy. I had a case of whooping cough twenty years ago in which the child had very severe convulsions and a doctor called it epilepsy. At the time I did not concur in that diagnosis. The boy has never had an attack of convulsions since the whooping cough ceased, but that sword has always hung over the head of his mother, because one physician had pronounced the convulsions epileptic Very many of these cases of convulsions have some of the appearances of epilepsy, but I teach my students to be very careful about calling them epilepsy. One should never take away the last hope of the mother by telling her that her child is an idiot. In making a differential diagnosis I am very slow to pronounce a case one of epilepsy as very few children have convulsions after they have passed the period of infectious diseases. I have had children who every time they had an infectious disease had convulsions. In one family I had three children and in one of them every time the temperature went over 102 F. the child had convulsions, while in two brothers the temperature might go to 104 and 105 and there would be no convulsions.

DR. SHAW: There is a tendency both among members of the medical profession and the laity to think that once an epileptic, always an epileptic. The question of prognosis is a serious one, but I do not feel that we should give a hopeless forecast. I have had several cases of undoubted epilepsy in young children which have recovered, that is, the attacks never returned after a period of several years. Still quotes from Hippocrates, "They who are troubled with the falling sickness before they attain the age of 14 may be freed from it."

DR. HELMHOLZ: I have usually grouped the first three classes under spasmophilia. I have thought that acute conditions, particularly gastro-intestinal disturbances, may be an element necessary to change a latent spasmophilia into an active one.

In how many of the cases were the electric reactions taken?

It seems to me that the point made by Dr. Morse is of great importance, namely, that if there is a rise of temperature at the onset of an illness, convulsions associated with it are not of so great importance so far as the diagnosis of epilepsy is concerned as if there was no fever and other symptoms of an infection.

DR. TALBOT: If this symptom is in some instances due to spasmophilia, one would expect to find some change in the blood calcium. We have determined the blood calcium in a small number of spasmophiliacs and found the blood calcium lowered. In considering this whole question emphasis should be laid on the fact that we are dealing with a symptom and not with a disease.

DR. MORSE: I have not considered breath-holding as a symptom of spasmophilia. I think that long, hard labors to be considered just as dangerous as operative labors. As to the cases of whooping cough and birth hemorrhage, I have left out all cases in which there were any signs of injury to the brain, and when there are no signs of injury, how are we going to know whether there is hemorrhage or not. I have not seen convulsions in whooping cough as often as Dr. Holt has. In my experience the great majority of patients with whooping cough with convulsions have died of bronchopneumonia or hemorrhage.

We see any number of cases at the hospital for diagnosis as to whether they have epilepsy or not. If they have had convulsions for any length of time we do a lumbar puncture, a Wassermann test, examine the stools and the urine, have the fundi examined, and give the child a thorough physical examination. In these cases we seldom find anything to account for the convulsions, and after all our efforts are no more able to tell whether they have epilepsy or not than before it began. Only the other day we had a child that was examined in this way three different times and as no cause for the convulsions was found, a diagnosis of epilepsy was made. The child's mother reported that an old lady gave him some worm medicine which caused the expulsion of several round worms, and he had not had a convulsion since.

As to the question whether these older children may have spasmophilia, I do not know. It does not seem probable that when a child has a convulsion once in six months or a year that he has spasmophilia all that time and yet it only shows itself so rarely.

A RÉSUMÉ OF SOME EXPERIMENTAL STUDIES ON CUTANEOUS HYPERSENSITIVENESS

E. C. FLEISCHNER, K. F. MEYER

AND

E. B. SHAW

SAN FRANCISCO

About ten years have passed since Mantoux and Moussu[1] in France, and Mendel[2] in Germany, first called attention to the value of the intradermal method of eliciting cutaneous hypersensitiveness, although in 1903 Mendel established the fact that, if in giving tuberculin intravenously a portion went into the tissues around the vein, a severe reaction took place in infected individuals. Antedating these observations by several years, however, Escherich described a local reaction following the subcutaneous injection of tuberculin, which he called "stich reaktion," and in 1905, von Pirquet[3] published his epoch making observations on cutaneous hypersensitiveness, to which he applied the term "allergy." Since these publications, it is, indeed, striking to note the paucity of investigators that have interested themselves in this fascinating problem. A fairly large number of men has drawn clinical deductions on the relative value of the intradermal test, as compared with the von Pirquet reaction, but work tending to explain the cause or the meaning of cutaneous hypersensitiveness has been conspicuously rare. Furthermore, it is singularly striking that whereas the original experimenters based their results entirely on findings in animals and humans suffering from tuberculosis, it has not stimulated others to study the question in relation to other diseases.

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* From the George Williams Hooper Foundation for Medical Research and the Pediatric Department of the University of California Medical School.

1. Mantoux, C., and Moussu, G.: Compt. rend. Acad. d. sc. **147**:592, 1908.
2. Mendel, F.: Med. Klin. **12**:402, 1908.
3. Von Pirquet: Handb. d. Technik & Methodik d. Immunitätsforschung, 1911, Suppl. 1, p. 191.

In Europe Römer[4] investigated the cutaneous tuberculin reaction most intensively, and in this country, Baldwin[5] and later Krause[6] studied the subject, particularly in experimental animals, drawing from their work many valuable conclusions. In an effort to determine, if possible, the cause of cutaneous hypersensitiveness, Baldwin placed in the abdominal cavities of guinea-pigs, capsules of porous Berkefeld filter clay, some of which had been filled with living tubercle bacilli, and others with filtered watery extracts of bacilli, i. e., tuberculoprotein, and then sealed them with dental cement. Cutaneous hypersensitiveness was not present in tests made on the fifteenth and fifty-seventh day after the experiment. The capsules became encysted in a few weeks and Baldwin drew the conclusion that cutaneous hypersensitiveness did not develop through the mere presence of tubercle bacilli in the body, even though they were more or less accessible to the body fluids. Anaphylaxis was present in these animals. Believing that absorption from the capsules might be either too quick or not sufficiently prolonged, he used an emulsion of animal charcoal that had absorbed the bacillary extract, and injected it under the skin and into the peritoneal cavities of his animals. The results were likewise negative on the twenty-first, one hundred and sixth, one hundred and fifty-fifth and two hundred and sixth day. Conjecturing that cutaneous hypersensitiveness depended on some property inherent in the serum of tuberculous animals, he injected into the guinea-pigs which had received the charcoal emulsion of bacillary extracts, serum of diseased pigs before and at the time of the tests. His results were entirely negative.

Assuming the possibility that the fatty or waxy substances in the bacilli might have something to do with the development of positive tests, he injected small amounts of benzol extracts of tubercle bacilli intraperitoneally, and alcohol ether extracts subcutaneously and intraperitoneally into animals that had already received the protein in charcoal. Positive tests were conspicuously absent. He was not able to produce cutaneous hypersensitiveness by injecting either the whole blood or the serum of hypersensitive guinea-pigs. He was likewise

4. Römer, R. H., and Joseph, K.: Beitr. z. Klin. d. Tuberk. **14**:1, 1909.
5. Baldwin, E. R.: J. Nat. Assoc. Study & Prev. Tuberc., 7th Ann Meeting, 1911, p. 351.

unable to get positive tests when he used for testing an antigen made from the plasma of tuberculous livers, spleens, glands, old or new tubercles.

Baldwin therefore concludes that for positive cutaneous hypersensitiveness to be present, tubercle must exist in the body. These tubercles may come from the injection of dead sterilized bacilli, in which case the positive reactions are less persistent than in animals subject to living infection. He states further that the persistency and delicacy of the reaction appears to be associated with tubercles which are not wholly fibrous. It may be due to slow disintegration and digestion of the bacilli in the periphery of the caseated center. Baldwin adds that he can understand why such a reaction should occur in the focus of infection, because of changed cell functions created to meet the need at the time and place, but how the distant cells in the skin can acquire these reacting functions, is less easily comprehended. His conclusion, therefore, seems warranted that in nature's method of extracting and disintegrating bacilli, some product is set free in the blood to act on the distant cell groups not connected with the tubercle, or in contact with the bacilli.

Following Baldwin's very valuable work, Krause[6] carried out a series of experiments having important bearing on the problem. He injected guinea-pigs with two well known strains of tubercle bacilli, one of which, H37, was very virulent, causing generalized tuberculosis, the other, R1, was very benign, with a tendency simply to involve regional lymph nodes, from which process the animals ultimately made a complete recovery. The group injected with H37, the virulent strain, showed increasing cutaneous hypersensitiveness from eleven days after the injection, when the first regional changes were noted, to the forty-sixth day, when the disease was well advanced. At every stage the reaction was more intense than in the R1 animals. In the group injected with the R1 strain, the reaction increased as the disease developed. After it came to a standstill and began to heal, the cutaneous hypersensitiveness became milder. The ability to react, however, never entirely disappeared.

Krause raises the question from this observation, as to whether an infection like tuberculosis, once it gains anatomic (not mere bacillary) foothold, is ever entirely wiped out. In the guinea-pigs injected

6. Krause, A. K.: J. M. Res. **35**:1, 25, 43 (Sept.) 1916.

with the R1 strain, he often found no tubercles macroscopically, but was able to demonstrate small ones microscopically, so that there is always the possibility that a tubercle, however small, may exist to give cutaneous hypersensitiveness.

To determine the effect of reinfection on the power to react cutaneously, Krause injected animals that were recovering from the R1 strain with the virulent organism, H37. Cutaneous hypersensitiveness rapidly increased. He also injected a set of guinea-pigs with acid fast Timothy bacilli, which produce a tumor going on to abscess formation from two to four days after inoculation, with regional lymph node enlargement. Seven days afterward he got positive skin tests, which had diminished very much on the twenty-second day. Twenty-five days after the original infection, the animals were again injected with Timothy bacilli. Three days later, twenty-eight days after the first injection, the skin tests were more pronounced than at any time, confirming the observations made with the tuberculous animals, that reinfection with an organism increases cutaneous hypersensitiveness. Timothy bacilli produce lesions in guinea-pigs very much more rapidly than tubercle bacilli, and corresponding to this, positive skin tests appear much earlier. Both lesions and hypersensitiveness, however, are more transitory in Timothy bacillus disease than in tuberculosis.

Krause injected a series of animals that had been infected with the virulent H37 organisms and had developed cutaneous hypersensitiveness, with tuberculin intraperitoneally. They became very sick, and during this period, the skin tests, which had been strongly positive, diminished very much in intensity. His conclusions are: 1. Cutaneous hypersensitiveness is never present without a focus. 2. It appears coincidental to the establishment of the focus. 3. It diminishes with the healing of the focus. 4. It varies with the virulence of the invader. 5. It is probably never entirely lost except in the presence of pregnancy or intercurrent disease. 6. It is increased by reinfection. 7. It is diminished or completely wiped out during a period of general tuberculin reaction. 8. The suggestion is offered that tissue hypersensitiveness may be a function of immunity to reinfection.

Gelien and Hamman,[7] who made a large number of observations on cutaneous hypersensitiveness in humans, conclude that, since nearly all

7. Gelien, T., and Hamman, L.: Bull. Johns Hopkins Hosp. **28**:163 (May) 1917.

adults are infected with tubercle bacilli, one may assume that a low grade of tuberculin hypersensitiveness is constantly present. With a fresh invasion, either from within or without, this increases. If the disease subsides and the individual recovers, the hypersensitiveness falls to a lower or the original level. If the tuberculosis remains active, the high level of the hypersensitiveness persists and lasts until the body is overwhelmed and the resistance broken entirely down by the disease, at which time all evidence of cutaneous hypersensitiveness ceases.

It is apparent, therefore, that these investigators explain cutaneous hypersensitiveness on the assumption that it is always an evidence of infection, and their experiments are ample proof of their contention.

During the past three years we have made an intensive study of cutaneous hypersensitiveness in connection with several problems that entailed the infection of a large number of guinea-pigs. In 1916, certified milk was studied in an effort to determine the presence of tubercle bacilli, and incidently an enormous number of animals developed the lesions produced by the *B. abortus bovinus.* In the diagnosis of these infections, no test was so constantly positive as the intradermal skin test. During the same period, many guinea-pigs were infected with tuberculous material from various sources, and positive intradermal reactions were always and only present in the diseased pigs, so that the conclusion was drawn that in guinea-pigs which are infected with *B. abortus bovinus* or *B. tuberculosis,* positive cutaneous hypersensitiveness is always an indication of infection.

During 1917, the problem was attacked from another angle. It seemed highly desirable, after contending that positive cutaneous hypersensitiveness was always an evidence of infection in guinea-pigs, to immunize a series of animals by the repeated injection of antigens derived from the organisms with which the preceding guinea-pigs had been infected, and to study the skin reactions in these immunized pigs. Gay and his co-workers[8] had previously claimed that positive skin tests were an evidence of immunity, drawing their deductions from work with typhoidin. Nichols[9] had questioned this conclusion (?), however, because he obtained similar typhoidin reaction in soldiers who had been vaccinated with paratyphoid, and it is well known that para-

8. Gay, F. P.: Arch. Int. Med. **13**:471 (May) 1914; **14**:671 (Nov.) 1914.
9. Nichols, H. J.: J. Exper. M. **22**:78, 1915.

typhoid vaccination will not immunize against typhoid. He considered the reaction an evidence of previous protein sensitization. Austrian,[10] Kolmer[11] and many others likewise failed to get results whereby the work of Gay and his pupils could be authenticated. Meyer and Christiansen[12] failed to confirm the observations of Gay by conducting a very searching inquiry into the mechanism of cutaneous hypersensitiveness in the rabbit. A positive typhoidin reaction in a rabbit never indicated, according to their observations, that this animal will resist a subsequent intravenous injection of living typhoid bacilli, or that the animal is so protected that it will not become a chronic carrier of bacilli in its gall-bladder or liver. Unpublished experiments with other organisms than the typhoid bacillus confirmed these conclusions, and also convinced these workers that the rabbit is a most unsatisfactory animal for the study of cutaneous hypersensitiveness.

Bearing this rather conflicting literature in mind, guinea-pigs were immunized by repeated intraperitoneal injections of the following antigens: dead *B. abortus bovinus,* dead *B. typhosus,* old tuberculin, bovine, *B. abortus protein.* That a high grade of immunization was established in all of these animals was evidenced by the constantly positive agglutination reactions. The antibody content of the blood must have been very great, because the tests were distinct in dilutions as high as 1:2,000 in the pigs that had been injected with all of the antigens except the tuberculin. Complement fixation tests on the serum of these pigs, using the tubercle bacillus as an antigen, were unsatisfactory, which corresponds to the experience of many investigators on complement fixation in tuberculosis. Intradermal tests were made on all of these animals, and in no single instance was a positive one obtained. Postmortem examinations failed to reveal any evidence of infection.

Not content with drawing a conclusion from this set of experiments, another series of animals was injected with the same antigens, using much larger dosage. Besides repeating the above mentioned injections, one set of ten pigs was injected with dead rodent *B. paratyphosus,* that was known to be very virulent for guinea-pigs. Agglu-

10. Austrian, C. R., and Bloomfield, A. L.: Arch. Int. Med. **17**:663 (May) 1916.

11. Kolmer, J. A., and Berge, Jr., J. H.: J. Immunol. **1**:409 (Aug.) 1916.

12. Meyer, K. F., and Christiansen, C. R.: J. Infect. Dis. **20**:357, 391 and 424 (April) 1917.

tination tests were constantly positive, cutaneous hypersensitiveness was always negative and necropsies failed to show the presence of the slightest lesion in any animal. It seemed, therefore, justifiable to state that while in guinea-pigs, cutaneous hypersensitiveness was always a sign of infection, a high state of developed immunity without infection evidenced by a pronounced antibody content of the blood, giving positive agglutination reactions in high dilutions, never produced cutaneous hypersensitiveness. Stated simply, in guinea-pigs cutaneous hypersensitiveness is always a sign of infection and never a sign of immunity.

These experiments establish for the guinea-pigs a definite biologic law. Conditions which will sensitize an animal in an anaphylactic sense, will not necessarily sensitize its skin, and that to bring about cutaneous hypersensitiveness, certain very definite and strict requirements must be fulfilled. In no other way than by the introduction of bacilli and the consequent production of foci has anybody thus far succeeded in sensitizing a guinea-pig's skin. The mere injection of protein, soluble or insoluble, will not produce skin sensitiveness, but will render the animal anaphylactically sensitive.

During the past year, this work has been continued, using a variety of infecting organisms for confirmation of previous results. Furthermore, it seemed essential to study the problem from other angles. Baldwin, in his work, had made the statement that anaphylaxis was present in his animals, although he did not obtain positive skin tests in the absence of infection. He did not elaborate, however, on the possible connection of these phenomena. Some observers, particularly Kolmer,[13] having contended that hypersensitiveness was an indication of the anaphylactic state, as it affected the skin, the importance of studying the relationship of cutaneous hypersensitiveness to anaphylaxis naturally suggested itself. Having demonstrated that animals that had been injected with dead organisms developed high antibody content in their blood, but never showed positive intradermal reactions, it seemed logical to study also the effect of inoculating these pigs later with living bacteria, and observing the result of this subsequent infection on the cutaneous hypersensitiveness.

While this work was in progress, Evans[14] published her very illuminating article, in which she showed that morphologically, sero-

13. Kolmer, J. A.: Bull. Johns Hopkins Hosp. **28**:163 (May) 1917.
14. Evans, A.: J. Infect. Dis. **22**:580 (June) 1918.

logically and biochemically the *B. abortus bovinus* and the organism which causes undulant or Malta fever, were practically indistinguishable. Her attention was first called to the problem by the observation made in this country that practically all raw cow's milk contained *B. abortus,* and the work of the British Commission, which showed that the milk of 41 per cent. of the goats in Malta contained *B. melitensis.* In her work, Evans used strains of *B. abortus* that were agglutinated by an abortus immune cow's serum in dilution of 1 : 1,280 or higher. She used six strains of *B. melitensis,* the history of five of which is as follows: (1) *B. melitensis* (Dr. Annett) was obtained from England in 1907; (2) *B. melitensis,* stock 22; (3) *B. melitensis,* stock, received from the Pasteur Institute in Paris prior to 1900; (4) *B. melitensis,* R. A. M. C., obtained from the Royal Army Medical College of London, England, in January, 1908; (5) *B. melitensis,* U. S. Navy, obtained from the U. S. Naval Medical School in March, 1909. These five strains responded alike to cultural, biochemical and serologic tests. Comparison of the *B. abortus* and the *B. melitensis* showed them to be identical as far as growth on the various culture media was concerned for the first seven days. After that, the *B. melitensis* growth was a little browner. The biochemical changes of the two organisms were the same, and the agglutination reactions to a *B. abortus antiserum* were exactly alike.

A series of animal experiments was carried out as follows: Four pregnant guinea-pigs were infected with *B. abortus* and four with *B. melitensis,* the method of inoculation not being stated. In a few days, three animals in each group aborted, and five days after the inoculation, one animal was killed from each set. In from three to four days, characteristic dew drop colonies made their appearance on both sets of slopes inoculated from the liver, spleen and uterus of the infected animal. Several weeks later, the *B. melitensis* slopes showed a more intense brownish coloring. No other difference was distinguishable. Thirteen days after the inoculation, serum was obtained for agglutination tests. The results with the guinea-pig *B. abortus* antiserum were the same as with the cow's antiserum. With the guinea-pig *B. melitensis* antiserum, however, the results were less uniform. *B. abortus* strains were agglutinated in dilutions of 1 : 320 and 1 : 640. Two of the *B. melitensis* strains were agglutinated in dilutions of 1 : 1,280, one 1 : 2,560, and the homologous strain in a dilution of 1 : 5,120.

Evans carried out an interesting set of absorption tests. For practical purposes using a *B. abortus* antiserum, this method cannot distinguish *B. abortus* from *B. melitensis.* Using a *B. melitensis* antiserum, the results are not quite the same. The melitensis antiserum acts on the melitensis antigen the same as the abortus antiserum acts on both. The melitensis antiserum acts on the abortus antigen the same as the abortus antiserum, except in characteristic lower dilutions. The melitensis serum with the melitensis antigen absorbs all the agglutinins, which are active toward *B. abortus.* Melitensis antiserum, after acting on *B. abortus,* acts in the same way on the melitensis antigen as it would have acted without being saturated. Evans explains these absorption tests by assuming that both the abortus and melitensis antiserums are alike in kind, but differ in proportion, and that the corresponding agglutinable substances are present in the bodies of the two species of bacteria in different proportions. One can only distinguish the two organisms by a *B. melitensis* antiserum, when the agglutinating strength of the serum for both species is known. Judging from these experiments, agglutination tests which have been relied on for the diagnosis of undulant fever have not proved whether the infections were due to *B. melitensis* or *B. abortus.*

In connection with a discussion of these absorption tests, it may be said that this method was used by Nègre and Raynaud[15] to distinguish *B. melitensis* from *B. paramelitensis* (the original strain of Bruce) based on the assumption that similar reactions had been considered sufficiently distinctive to separate *B. dysentericus* from *B. paradysentericus,* and the meningococcus from the parameningococcus. If their contentions are correct, Evans' work suggests that *B. melitensis* and *B. abortus* are more closely related than *B. melitensis* and *B. paramelitensis.* Incidentally, without at this time further elaborating on the possible pathogenicity for man of the *B. abortus,* one might call attention to a case of paramelitensis fever reported by Basset-Smith[16] in which the patient had all the clinical signs of undulant fever for two years, but never gave positive serologic reactions until the *B. paramelitensis* was used as antigen, when the tests were positive in high dilutions.

15. Nègre, L., and Raynaud: Compt. rend. Soc. de biol. **72**:664, 791, 1912.
16. Basset-Smith, P. W.: J. Roy. Naval Med. Serv. **1**:166, 1915.

Evans' results suggested two very fascinating possibilities, both of tremendous scientific interests and one of possible serious import. The first possibility was that in the *B. melitensis,* another organism, very intimately related to the *B. abortus bovinus* was obtainable, that was pathogenic for guinea-pigs, and might offer a very fertile field for the further study of cutaneous hypersensitiveness. The second possibility entailed a careful investigation of the pathogenicity for man of the *B. abortus bovinus,* considering its very great serological, biochemical and cultural similarity to the *B. melitensis,* the recognized and accepted etiologic agent in undulant fever. It is with the first of these problems, only, that part of this paper has to deal.

A careful résumé of the literature, with reference to guinea-pig inoculation with *B. melitensis,* showed a relatively small number of observations. Durham,[17] in 1898, injected guinea-pigs intracerebrally with cultures whose virulence had been increased by previous intracerebral inoculations, a method which had originally been used to raise the virulence of *B. influenzae.* In animals that died the organism was recovered from the viscera. Not much is given as to his post-mortem findings. In guinea-pigs that died at remote periods, the kidneys were intensely engorged, and in two instances the spleen was much enlarged, the other organs showing nothing abnormal. Following the intraperitoneal injections, the results were much more chronic in character.

In 1904, Carbone,[8] who later died of undulant fever contracted in his laboratory, showed that male guinea-pigs often developed a purulent inflammation of the tunica vaginalis, following the intraperitoneal injection of *B. melitensis.* It is to Eyre,[19] however, that most credit is due for having studied the *B. melitensis* in relation to guinea-pig infections. He showed that following the first isolation from human cases the *B. melitensis* could only kill guinea-pigs when given subcutaneously, intravenously or intraperitoneally in tremendous doses. Infection was also possible by intracranial inoculation. Using a freshly isolated strain, the infection was very chronic, the animals living from one to two hundred days. By repeated intracranial injections, he was able to increase the virulence of the organisms so that the pigs died within

17. Durham, H. E.: J. Path. & Bacteriol. **5**:377, 1899.
18. Carbone, T.: Arch. per le sc. med. **28**:273, 1904.
19. Eyre, J. W. H.: Report Mediterranean Fever Commission, 1905, Part 2, p. 67.

twenty-four to seventy-two hours after an intravenous injection three to twelve days after an intraperitoneal inoculation, and nine to thirty-six days after an intracranial inoculation.

Eyre described two types of infection in guinea-pigs, the acute and chronic, as follows:

Acute Melitensis Infection.—"Several days after a moderate dose of a highly virulent culture, or a heavy dose of a less virulent culture, the animal dies. For from two to three hours after the injection he seems to be all right and eats well, but loss of weight sets in, which is highly characteristic for the disease. At first the temperature drops rapidly, then rises. The animal sits huddled up, will not eat, becomes weaker, and if disturbed, goes into tonic convulsions. This stage of increased irritability goes gradually into a stage of coma, from which the animal can be aroused with difficulty, and in which he remains about twenty-four hours with a subnormal temperature. Sometimes he has a convulsion before death, but usually it occurs without warning. The postmortem findings are those of acute septicemia. The organisms can be recovered from all the tissues and organs. In male animals, following intraperitoneal infections, there develops a bilateral purulent process in the tunica vaginalis with beginning hypertrophy of the testes. This pus contains numerous organisms."

Chronic Melitensis Infection.—"With small doses of virulent, or fair doses of moderately virulent organisms, the animal gets a chronic infection. Aside from a progressive loss of weight and a severe anemia, there are no characteristic symptoms. Even the temperature is not much changed. The incubation period of the chronic variety of the disease is from two to three days. Then comes a time, three to six days, when the animal is perceptibly sick. He will not eat, stays in a corner of his cage, loses weight and is very weak. He gradually improves, eats well, even ravenously, and although losing no more weight, he never regains his original weight. Following weeks or months, during which, aside from the loss of weight, the animal seems well, he suddenly dies. Postmortem cultures from the liver are sterile; from the spleen and bone marrow there is sometimes a scanty growth, although these are also often sterile. From the kidneys and the urine a good growth of *B. melitensis* is obtained."

Nicolle and Conseil[20] report on two guinea-pigs which had naturally acquired a *B. melitensis* infection. One animal showed an enlarged spleen. And, again, in their subcutaneous inoculation and feeding experiments they record the findings of a hypertrophic spleen in some of the guinea-pigs examined.

The experiments on which this piece of work is based are divided into two groups. In the first group are included those observations that have a bearing on the relationship of anaphylaxis to cutaneous hypersensitiveness, and of cutaneous hypersensitiveness to immunity and subsequent infection. In the second group are included those experiments that pertain to the pathogenicity of the *B. melitensis* for guinea-pigs, and its relationship to the *B. abortus bovinus.*

In the carrying out of these experiments, that had for their object a study of the relationship of cutaneous hypersensitiveness to anaphylaxis, immunity and infection, the following method was employed The animals were immunized intensively by the repeated injection of various antigens, the high state of immunity being judged by the agglutination reactions. Intradermal tests were made upon these pigs, the antigen for this purpose being prepared as described in a previous paper. Certain of the animals were then selected for anaphylactic reactions. The proteins used for these tests were prepared, with the exception of the tubercle protein, in which the method described by Austrian[21] was followed, according to the procedure outlined by Zinsser and Parker.[22] After the lapse of a considerable period of time, some of the guinea-pigs were infected with living organisms corresponding to the antigens with which they had been previously immunized, and again cutaneous hypersensitiveness and anaphylaxis were studied.

Those experiments undertaken to determine the pathogenicity of the *B. melitensis* for guinea-pigs, and its relationship to the *B. abortus bovinus,* were carried out as follows: The infecting organisms were injected by the intraperitoneal and intratesticular routes. Twenty-five days after the injection, cutaneous hypersensitiveness was studied, and this was repeated on the fifty-fifth day, using as antigens melitensin, abortin, bronchisepticin and typhoidin. Agglutination tests were made,

20. Nicolle, C., and Conseil, E.: Compt. rend. Soc. de biol. **66**:503, 1909; **67**:267, 1909.

21. Austrian, C. R.: Bull. Johns Hopkins Hosp. **24**:141, 1913.

22. Zinsser, H., and Parker, J. T.: J. Exper. M. **26**:411 (Sept.) 1917.

using the *B. abortus bovinus* and the *B. melitensis* for comparison. The animals were later sacrificed, and careful observations carried out as to gross and microscopic pathology, after the viscera had been cultured to recover, if possible, the invading organisms.

The details of these experiments will not be given at the present time, but will be tabulated carefully and published later.

Careful study of the available data reveals some very interesting facts. By the repeated injection of dead *B. abortus* or abortus protein into guinea-pigs, it is possible to develop a high grade of immunity, as evidenced by the marked agglutinin content of the blood. Incidental to the development of this immunity, these animals became highly sensitized and some of them developed a pronounced anaphylactic state. Cutaneous hypersensitiveness is never present. By subsequently injecting them with living organisms, skin tests are always positive, if on postmortem examinations there is pathologic evidence of infection. Anaphylaxis persists, although its symptoms are neither more nor less pronounced in animals inoculated with dead or living bacteria. That there is an important individual predisposition present in the development of anaphylaxis is proven by the fact that certain guinea-pigs never show symptoms of the anaphylactic state, either after being immunized to or infected with an organism. It is possible to develop an anaphylactic reaction in certain of the guinea-pigs that are injected either intraperitoneally with heat killed typhoid bacilli, intravenously with heat killed typhoid bacilli or intraperitoneally with living typhoid bacilli. In none of these animals is cutaneous hypersensitiveness present. By subsequently injecting these animals with living typhoid bacilli intravenously, either sensitized or unsensitized, the skin tests become positive for a shorter or longer period, the extent of the reaction depending on the dose and the severity of the disease resulting in the guinea-pigs.

It is not within the scope of this paper to theorize on the development of positive skin tests in those animals that are inoculated intravenously with living typhoid bacilli. As far as is known, typhoid bacilli are not pathogenic for guinea-pigs in the sense that they are capable of producing a definite disease with visible anatomical lesions. And again the skin tests were frequently positive when cultural tests failed to reveal the presence of living typhoid bacilli. Whether the hypersensitiveness is a phenomenon depending on the changes elicited in the fixed tissues of the skin or on microscopic foci in the organs

following the intravenous injection of living typhoid bacilli, is something that only the future can decide. At any rate, one is justified in stating that it is possible to sensitize guinea-pigs to dead typhoid bacilli and have them highly anaphylactic without the development of cutaneous hypersensitiveness.

The lessons taught by the experiments with the rodent paratyphoid bacillus, which produces in guinea-pigs a distinct disease, characterized by pseudotuberculosis lesions, necroses in the liver and spleen, are much more definite. It is very readily possible to sensitize these animals by the subcutaneous and intraperitoneal injection of heat killed or tricresol killed paratyphoid bacilli without ever developing positive intradermal tests, although certain of these pigs are highly anaphylactic. Subsequently by feeding living organisms, infection takes place as proven by recovery of living bacteria from the feces and postmortem lesions, and the skin tests are invariably positive. The anaphylactic phenomena in animals previously sensitized are neither increased nor diminished following infection.

The importance of the experimental data gathered from the intratesticular injections of *B. melitensis* and the comparison of this organism with the *B. abortus bovinus* can scarcely be overestimated. Serologic and cultural tests previously made by Evans had clearly demonstrated that these bacteria were very closely related. Durham, Carbone, Eyre and Nicolle and Conseil had shown that the *B. melitensis* was pathogenic for guinea-pigs, but their descriptions of the pathology of the disease were wholly unsatisfactory. Following the intratesticular injection of certain living *B. melitensis* strains, lesions develop in all of the viscera, which are macroscopically and microscopically absolutely indistinguishable from changes produced in guinea-pigs by the *B. abortus*. Cutaneous hypersensitiveness is positive whether the *B. melitensis* or *B. abortus* is used as an antigen. Likewise if guinea-pigs are infected with abortus bacilli, skin tests are as strongly positive to melitensin as to abortin, although all other bacterial proteins give negative results. The serologic reactions are likewise positive using melitensis antiserum or abortus antiserum with *B. abortus* or *B. melitensis*. It is in this connection, however, in these experimental infections in guinea-pigs that one means is at hand for determining the infecting agent. The serum of animals infected with *B. melitensis* will agglutinate this bacterium in higher dilutions than the *B. abortus*, although the reaction with the latter organism is definitely positive.

These observations logically suggested some experiments on monkeys. As far as a survey of the literature can determine, only Fabyan[23] states that he has injected monkeys with the *B. abortus,* and that he was able to isolate the bacillus from the spleen (?) twelve and thirteen weeks respectively after the infection. The mode of infection, the clinical and serological response to the injected organisms has unfortunately not been stated. Interest naturally centered in discovering whether or not the *B. abortus* produces a temperature reaction similar to the one seen in apes infected with *B. melitensis.* Thus far only the following experiment has been concluded:

EXPERIMENT 1.—Injected macasus rhesus monkey intravenously with 1/10 twenty-four hour slant *B. abortus,* strain 80, April 4, 1919, after he had been under observation for several weeks to be certain that he had a normal temperature. Before injecting, he was bled for agglutination tests, which were negative to *B. abortus* and *B. melitensis.* The animal began to have an irregular temperature on the third day after the inoculation, which persisted up to the time he was chloroformed. On the eighteenth day, agglutination tests were positive with the *B. abortus* in dilution of 1:2,000, *B. melitensis* 1:1,000; on the twenty-fifth day, *B. abortus,* 1:2,000 to 1:4,000, *B. melitensis,* 1:800; on the thirty-fourth day *B. abortus,* 1:600; *B. melitensis,* 1:100; on the fiftieth day *B. abortus,* 1:1,000; *B. melitensis,* 1:600. The animal was sacrificed on the fiftieth day and the spleen, liver, kidneys and mesenteric lymph nodes were found to contain numerous *B. abortus bovinus.* Several skin tests with abortin and melitensin were negative. Microscopically, we failed to find characteristic cellular proliferation.

The infection of this macasus monkey by the intravenous injection of *B. abortus bovinus* is clearly indicated by the febrile course of the disease, the positive agglutination reactions, and the recovery of the offending organism from the viscera postmortem. Further study will be necessary, however, before definite information can be obtained as to the bearing of such a procedure on the possible pathogenicity of the *B. abortus* for man.

CONCLUSIONS

1. Cutaneous hypersensitiveness in guinea-pigs that have been inoculated with *B. tuberculosis, B. abortus bovinus, B. melitensis* or rodent paratyphoid is only positive in the presence of infection.

2. Following intravenous injection of living typhoid bacilli, temporary cutaneous hypersensitiveness can be produced frequently.

3. Guinea-pigs showing a high grade of acquired immunity to an organism, as evidenced by a strongly positive agglutination, comple-

23. Fabyan, M.: J. M. Res. **28**:82, 1913.

ment fixation reactions and complete resistance to a subsequent infection with living organisms, will never give specific positive cutaneous hypersensitiveness.

4. Bacterial proteins, soluble or insoluble, which sensitize a guinea-pig in an anaphylactic sense, will not sensitize its skin. A state of an anaphylactic hypersensitiveness can exist without the least cutaneous hypersensitiveness.

5. The *B. abortus* and *B. melitensis* are very closely related. Guinea-pigs infected by the *B. abortus* give just as strong a skin reaction with melitensin as do guinea-pigs infected with certain strains of *B. melitensis*. From the standpoint of bacterial protein sensitization, the relationship of these two organisms is closer than the one generally recognized between the *B. typhosus* and *B. paratyphosus*.

It is not unlikely that the *B. melitensis* is the caprine strain of the bovine *B. abortus*.

Studies on monkeys, goats and other animals to elucidate this very interesting question are in progress.

DISCUSSION

Dr. Cowie: I have been very much interested in this beautiful piece of work. I would like to ask Dr. Fleischner whether he has carried out observations on the offspring of animals with this disease and whether they too were hypersensitive.

QUARANTINE AND DISINFECTION IN SCARLET FEVER*

J. C. GITTINGS, M.D.
Lieutenant Colonel, M. C., U. S. A.; Chief of Medical Service, U. S. Army General Hospital No. 9, Lakewood, New Jersey
PHILADELPHIA

The first assignment of patients to the U. S. Army General Hospital No. 9 consisted of 139 patients with scarlet fever transferred in February, 1918, to relieve the pressure in the contagious wards of a nearby camp. The assignment was unexpected and the capacity of the ward set aside for contagious diseases was entirely inadequate, so that two additional wards of a capacity of ninety beds each were hastily converted into contagious wards.

The hospital functionates in a hotel of the orthodox double-L design, which is admirably suited for quarantining two entire sections. It was possible to secure absolute lack of contact between patients and the healthy, except in the inevitable instance when orders were not obeyed. The nurses on duty in the wards were isolated as to their sleeping quarters and mess. The ward attendants slept in a portion of the contagious wards but were allowed to mess together in the mess hall on the lower floor, after change of gown, cap and, presumably, disinfection of the hands.

The patients were admitted to the hospital in every stage of scarlet fever from the second to the thirty-fifth day, 20 per cent. being within the first five days of the disease. Opportunities for contagion therefore were numerous, and five additional cases which developed among contacts were treated in the wards. Two of the five occurred in assistant ward attendants after they had had one or two weeks' service in the infected wards; another occurrred in the brother of one of the ward attendants. This brother had been visited in his quarters by the ward attendant on the night before the latter was taken ill, and eight days later this brother developed a sore throat with slight temperature and ephemeral rash. As an isolated case it would undoubtedly not have been diagnosed. He was treated in the scarlet fever wards, however,

*Authority to publish granted by Board of Publication, Surgeon General's office.

without subsequent infection, and the conclusion was inevitable that the attack instanced a very mild type of scarlet fever of the kind most apt to spread contagion. The fourth patient was one of the kitchen detail who took meals to the ward. Contrary to orders, he entered the diet kitchen instead of passing in the food from the outside. He was taken sick the day after reaching his home on a furlough, and was not included in the total number of patients treated at this hospital. The fifth patient was taken ill six days after preparing a large number of infected blankets for sterilization. He had nursed through the epidemic and no particular caution was deemed necessary in assigning him to this work. The agency of the infected blanket in his infection is not positive but strongly suggested. The sixth patient, whom we will designate as patient B, was admitted to the surgical wards in another part of the building, suffering from tuberculous adenitis with an open sinus which required dressing, two days after quarantine had been lifted on the last of the original cases of scarlet fever. Seven days after admission he developed a sore throat followed by the rash on the next day. Patient B gave no history of direct exposure at the camp from which he was sent to the hospital and knew of only one case of the disease existing at that camp at the time he left it. The only possible exposure at this hospital was as follows: One of the original scarlet fever patients, whom we will call patient A, had developed a submaxillary adenitis on the forty-second day of his disease. This was at first supposed to be mumps, but in the course of twelve days suppuration occurred and on the fifty-fourth day of his disease he was transferred to the surgical ward for operation. The surgeons were informed of all the circumstances and agreed to accept the case under proper precautions. The abscess in the submaxillary gland in patient A was opened five days before the admission of patient B. Both patient A and patient B were dressed in the same dressing room, the surgeon stating that patient A was always dressed after every other case. Patient B states, however, that on at least one occasion, shortly after his admission, he was dressed immediately after patient A. It was impossible to verify this by other testimony and we must remain in doubt as to whether the infection of patient B resulted from any fault in technic or whether he had been infected prior to admission to this hospital. No other cases developed after patient B.

A uniform minimum length of quarantine of forty-two days from the day of entry into the camp hospital was enforced. On the fortieth

day each patient was carefully examined by the assistant chief of the medical service, Major William C. Moore. All those who showed any evidence of abnormality in the throat, nose or ears were inspected by the throat and nose specialist, Capt. Clarence Keeler, who decided whether or not the patients gave an evidence of being potential carriers of contagion. Those with disease of ears or upper air passages were given daily treatment. Fifty-nine per cent. of the patients were ready for discharge at the termination of the quarantine period, and 41 per cent. required more or less treatment. The average period of quarantine therefore was forty-seven days. This extension of time was due partly to the occurrence of several cases of mumps. Every patient exposed to this was kept in quarantine for the twenty-one days' period of incubation for mumps, unless he had been immunized by a previous attack. Only three cases of mumps developed, but the number of quarantine days was materially increased on account of this outbreak.

The construction of the hospital allowed space in the largest rooms for not more than six beds, while the average number of beds per room was only three. This type of construction increases the difficulties of nursing but unquestionably tends to prevent the spread of contagion. Many of the rooms had private baths adjoining them so that it was possible to effect complete isolation of contacts.

Upon the conclusion of quarantine, each patient was given an orthodox bath, shampoo, change of clothing, and was then transferred to a clean ward in another part of the building. All soiled linen was soaked in 2 per cent. liquor cresolis solution for two hours or longer before being sent to the laundry. Shoes, hats, and leather belts were exposed to formaldehyd gas in sealed closets for twenty-four hours. Blankets and patients' woolen clothing were sterilized in the autoclave. After the evacuation of the ward, the hall carpet and paper wadding were carefully rolled up and dropped from a window to the ground, then spread out in an unfrequented space in a corner of the hospital yard and left to air for forty-eight hours. After this the carpet was brushed and stored, ready for re-laying. (The use of carpets, of course, was absolutely unorthodox, but no other floor covering was available and some sound deadener in the halls was essential.) The floors of the wards and halls were mopped with a copious supply of the 2 per cent. solution of cresolis and all flat surfaces, bedsteads, furniture and wood work were wiped off with the same solution. The mattresses were left on the beds and every window in the ward was

left open for three days to the April winds and sunshine. The attempt was made to expose the pillows to concentrated formaldehyd gas, but, in view of the difficulty in securing surface contact and penetration, the attempt was abandoned and the pillows thereafter were thoroughly sunned and aired. At the conclusion of the airing the hall carpets were relaid and the wards equipped for use. Within two weeks they were again occupied and have been in constant use ever since. Until February, 1919, there had not been another case of scarlet fever observed in this hospital. Many of the patients discharged from the scarlet fever wards remained in the hospital for several weeks and had free access during pass hours to all parts of Lakewood and at times to neighboring towns on the coast. So far as can be learned none of these patients carried the contagion to anyone in Lakewood or elsewhere.

Certain statistical data may be added. The longest quarantine was sixty-three days and seventy-two days in patients suffering respectively from hypertrophiied tonsils, chronic follicular tonsillitis, severe deflection of the septum, and chronic nasopharyngitis, otitis media, chronic tonsillitis and hypertrophied tonsils.

Physical Defects.—Of the 144 patients, fifty-six exhibited no defects of any kind; ten exhibited defects which should have been a cause for rejection for military duty, viz., deafness (1), gastric ulcer (1), valvular heart disease (2), hysteria (1), chorea (1), Graves' disease (1), tuberculosis (3). Seventy-eight patients exhibited minor defects, seven of these consisting in simple enlargement of the thyroid without toxic symptoms; the other seventy-one had defects which were remediable, chiefly hypertrophied tonsils and dental caries.

Complications.—Eleven patients showed what might be called serious complications: pleurisy, otitis media, mastoid and cerebellar abscess, nephritis, myocarditis. The patient with mastoid infection and subsequent cerebellar abscess was the only one whose condition was at all critical. He also suffered from a toxic nephritis. His ultimate recovery was complete except for slight residual vertigo. Thirty-seven patients showed mild complications: transient albuminuria, bronchitis, functional cardiac disorder, pharyngitis, peritonsillar abscess, etc. Thirteen patients suffered from the symptom complex which has been described at this hospital as functional cardiac disorder, postinfectious type. All of these patients recovered before their final discharge from the hospital.

Length of Quarantine.—Until the infecting organism of scarlet fever is demonstrated, the proper length of quarantine always will be open to argument. In accounting for the spread of this disease, the chronic carrier would seem to be the most likely agent, as is known to be the case in diphtheria and meningitis. *A priori* the unhealthy nasopharynx and throat might be under the suspicion of harboring infection. Examination by a throat specialist therefore may enable us to prevent the discharge of a potential carrier and possibly to hasten the discharge of a healthy person. In mild cases, with healthy throats, thirty-five days or less might cover the danger period. The period of quarantine prescribed in the army is forty-two days, and under circumstances such as those just described, where discharged patients were to mingle at once with uninfected patients and with the towns people it was much better to err on the side of safety than to run any risk of a premature discharge. The isolation for such a long period was rendered much less irksome because of the opportunity for exercise through the well-ventilated corridor of a ward 300 feet long, and for recreation of various kinds.

Disinfection.—The factor of the human carrier in the etiology of contagious diseases is being accepted to such an extent as to make it extremely questionable whether any measure other than a thorough cleansing is necessary after any infection. Formaldehyd gas in proper concentration and with sufficient moisture probably will destroy the organism of scarlet fever. That this optimum result is often obtained is extremely doubtful. Disinfection of linen and woolens and disinfection and removal of the dust which accumulates on floors and flat surfaces, followed by thorough airing, appear to be the actual requirements for safety.

CONCLUSIONS

1. The human carrier is by far the most important factor in the transmission of the contagion of scarlet fever.

2. The actual limit of quarantine in uncomplicated cases probably may be placed safely at not more than thirty-five days; before quarantine is lifted an examination of the nose, ears, nasopharynx and throat by an expert should be the determining factor.

3. Thorough disinfection of linens and woolens with disinfection and removal of dust wherever it may have lodged, followed by thorough

airing, constitute sufficient protection after the contagion of scarlet fever and are surely more effective than disinfection by formaldehyd gas as it is usually practiced.

DISCUSSION

DR. KOPLIK: Patients with open wounds are peculiarly susceptible to scarlet fever and the same is true of burns. Patients subjected to operation should not come into contact with scarlet fever. Scarlet fever is not contagious from the epidermis, but it is from suppurating ears and glands. The important point is not contact with linen, clothes, etc., but with human carriers. Very rarely is infection due to clothing, but to contact. The disinfection of rooms is not as important as the disinfection of carriers.

With reference to the question of quarantine for six weeks and desquamation, the desquamation of the epidermis may last for months and months and this seems to have nothing to do with contagion. I do not think there can be a definite time limit for quarantine and at present the question is whether there is any focus of infection present on the skin, or when infection disappears from the epidermis.

DR. HERRMAN: In scarlet fever as in most of the common contagious diseases of childhood, the infectious material is conveyed by persons, not by things. Infection takes place from a true case, or from a recognized or unrecognized carrier. The vast majority of the cases which are cited in the literature as proving the possible conveyance through inanimate objects, will not bear a careful analysis. The desquamating scales play no part, the secretions and excretions carry the infectious material. The duration of quarantine will therefore depend on the persistence of these discharges, especially those from the nose and ears. The control of all carriers is practically impossible. The spread of those diseases in which carriers play an important rôle will only be possible when we have a method for the detection of susceptibles and a method of immunization of such susceptibles.

DR. MILLER: What is the length of time for quarantine in scarlet fever? There are those who believe that desquamation from the epidermis is not contagious, and if that is the case, what is to be our belief in regard to the duration of quarantine? Of course, we cannot keep the patient quarantined if desquamation continues indefinitely. The throat and general symptoms often disappear in mild cases after the acute stage, so that the duration of anginal symptoms cannot serve as a guide to the period of quarantine. Until we have more definite information, desquamation should be the ordinary test, and quarantine should be continued until desquamation is complete, i. e., ordinarily about five or six weeks.

DR. GRIFFITH: In the older literature as compared with the modern a very different idea prevailed in regard to the contagiousness of scarlet fever. One instance is on record in which some hair from a child dying from scarlet fever was preserved in a locket. Twenty years later another child found the locket and contracted the disease from the handling of the hair. This was fully believed in at that time, but we cannot accept such a view with our present knowledge of the disease. The idea still persists, however, in many quarters that rooms occupied by scarlet fever patients are for a long while to be held under suspicion. This seems to me to be unfounded. A recent bulletin of the department of health of Philadelphia speaks of their experience in this

matter. A ward which had previously been used exclusively for scarlet fever patients was filled with patients suffering from grippe during the recent epidemic. Only one case of scarlet fever developed, and there was no reason to think that that was contracted through the occupancy of the room. The prevailing belief is, I think, the correct one; that scarlet fever is transmitted by direct contact, and that the persistence of desquamation is not to be regarded as a criterion as to the duration of the infectiousness. The experience of the physicians to the English Fever Hospitals shows that from 2 to 4 per cent. of "return cases" occurred after a quarantine of six weeks, and that the number was not increased if the quarantine was reduced to four weeks, disregarding entirely the existence or nonexistence of desquamation. The harmfulness of basing the duration of quarantine on the presence of desquamation was well shown in one case under my care several years ago. After a quarantine lasting seven weeks I reported the case for discharge, but the health officer refused to permit of a discharge on the ground that the child was scaling. He changed his mind after he learned from me that the little boy was suffering from ichthyosis, and had been scaling and would scale all his life.

DR. HEIMAN: I am glad we are getting away from the time honored superstition that scarlet fever is carried by clothing. A person to acquire the disease must either come into contact with a patient having the disease, or with a carrier. The disease is conveyed more quickly by personal contact than by a healthy carrier. Other diseases are more easily conveyed by healthy carriers than by individuals affected with the disease. Formerly the period of quarantine in New York was forty days but it is now thirty days. Another point is in regard to susceptibility. The susceptibility in measles is from 90 to 95 per cent., while only 20 or 25 per cent. of children have a susceptibility for scarlet fever.

DR. KNOX: I should like to ask the writer of the paper whether he has any data as to how long patients who eventually develop scarlet fever continue to have sore throat with some malaise as the only symptoms and whether during this period they are infectious to others. I have seen several cases in which sore throat is the only symptom complained of ten days or two weeks before any eruption appeared on the skin and in one of these cases a small house epidemic occurred, the infection occurring before the first patient developed a rash. The observation of the infectiousness in the early stages in scarlet fever is much more important I believe than the isolation of patients until desquamation is complete, for I doubt if convalescent cases, even during active desquamation, are particularly infectious.

DR. GITTINGS: So far as we could observe, the period from the development of the sore throat to the appearance of the rash was from one to two days. This was in contradistinction to the cases observed during an epidemic this year, when many of the patients exhibited anginal symptoms for several days before the exanthem developed.

In the cases covered by the present report we paid no attention to desquamation other than to record its occurrence and it was not taken into account in determining the limits of quarantine. As to the length of quarantine I am inclined to believe that thirty days are sufficient for uncomplicated cases. An important practical factor is the character of the contacts which the convalescent will develop. If he is to mingle after quarantine with children a longer period than thirty days might be safer. If, on the other hand, he is to be in contact only with adults and with children immunized by a previous attack, thirty days should be sufficient.

THE CHOLESTEROL METABOLISM IN INFANTS*

KENNETH D. BLACKFAN AND JAMES L. GAMBLE

Recent animal feeding experiments have very clearly shown that the maintenance of the chemical structure of the tissue cells and their growth depends on the presence in the food of a number of substances, most of which are quite complex as regards their chemical structure. The amino-acids are an example of structural units which the food must contain. The requirements for these substances are absolutely specific. The absence of a single essential amino-acid from an otherwise adequate diet will interfere with tissue maintenance and prevent growth. Substances (fat soluble A, water soluble B and inorganic salts) other than the amino-acids have been shown also to be essential for tissue maintenance and for growth. Cholesterol, a complex substance, which is present in all of the tissues of the body, is an important structural unit of protoplasm, but very little is known regarding its relation to processes of metabolism. As cholesterol is present in the food of infants it seemed to us a matter of practical importance to determine quantitatively the intake and excretion. It is obvious that if it cannot be synthesized in the body, the necessary intake should be determined.

Several investigators have undertaken to demonstrate the synthesis of cholesterol in the animal organism by tissue analyses of growing animals on a cholesterol free diet. Gardner and Lander[1] using this plan of experiment decided that there was little if any synthesis of cholesterol in growing chicks. Dezani,[2] on the contrary, claims that growing mice on a cholesterol-free diet synthesize cholesterol readily. The results of these experiments are very questionable from the fact that plant foods contain substances known as phytosterols, which are nearly identical in their chemical structure with cholesterol. Ellis and Gardner[3] have shown that the cholesterol content of the blood of animals may be increased by feeding certain plant leaves which are rich in these phytosterols.

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* From the Department of Pediatrics, Johns Hopkins University and Harriet Lane Home, Johns Hopkins Hospital, Baltimore.

1. Gardner and Lander: Proc. Roy. Soc., Series B **87**:229, 1914.
2. Dezani, S.: Arch. di Farmacologia **17**:4, 1914.
3. Ellis and Gardner: Proc. of Roy. Soc., Series B. **85**:385, 1912.

In the food cholesterol is practically all free. In the tissues and in the blood it is found free and as ester in fairly constant proportions. It is excreted in the bile as free cholesterol. It is remarkably unique among the food substances in that it completes its metabolic cycle without undergoing any disintegrating change. As it is excreted into the intestine it can be used over and over again. This probably occurs to a considerable extent. A large fraction of it is lost, however, in the feces, where it is changed by reduction to a substance known as coprosterol. Ellis and Gardner[4] measured by weight the coprosterol in the stools of normal adults and compared it with the estimated cholesterol content of the food. They decided that there was enough cholesterol in the food to account for the amount of coprosterol found. Mueller,[5] in 1900, reported that the stools of typhoid patients on a milk diet contained mostly cholesterol and very little coprosterol. This led him to examine the stools of an infant. He found little or no coprosterol present. This observation of Mueller's suggested to us the possibility of studying very directly the question of cholesterol synthesis by comparing quantitatively the intake and excretion in infants.

Quite simple and satisfactory colorimetric methods are now at hand. We were unable to use for determination of cholesterol in milk and stool samples the direct method of Bloor.[6] The color obtained from the extract by this method was usually so different in quality from that of the standard that even a rough comparison was impossible. In these experiments we have used a modification of the Autenreith and Funk method,[7] which has been checked in a few instances by the gravimetric digitonin method of Windaus.[8] That we

4. Ellis and Gardner: Proc. of Roy. Soc., Series B. **86**:13, 1913.

5. Mueller, P.: Ztschr. f. physiol. Chem. **29**:129, 1900. Windans, A.: Ztschr. f. physiol. Chem. **65**:110, 1910.

6. Bloor, J.: Biol. Chem. **17**:4, 1914, 1916.

7. Autenrieth, W., and Funk, A.: München. med. Wchnschr. **60**:1243, 1913.

8. Method used for cholesterol determinations:

The samples of milk or of dried stool were digested for two hours on the steam bath with 15 to 20 c.c. of 30 per cent. potassium hydroxid. (The removal of cholesterol from the digests by shaking out six times with ordinary ether as in the method of Autenreith and Funk from similarly prepared digests of blood samples was found to be very tedious, and usually unsatisfactory because of the frequent formation of an emulsion. We found that we could apply very successfully at this point the plan devised by Kumagawa and Suto (Biochem. Ztschr. **8**:212, 1908) for separation of nonsaponifiable material from soaps.) An equal volume of 95% alcohol was added to the digest in a shaking cylinder

were dealing with cholesterol and not coprosterol or similar substances which might give the same color as cholesterol was established by weighing the substance extracted from the feces. This was then dissolved in chloroform and compared in the colorimeter with that of a standard solution of pure cholesterol. In all instances, we obtained an agreement between the weight and color value quite within the range of experimental error. These results satisfactorily proved that the cholesterol excreted was unchanged in the feces and permitted us to compare quantitatively the cholesterol contained in the food with the amount excreted.

TABLE 1.—COMPARISON OF WEIGHT AND COLOR VALUE OF EXTRACTED MATERIAL SHOWING THAT CHOLESTEROL IS EXCRETED UNCHANGED IN THE FECES

Specimen Stool	Weight of Extract, Mg.	Color Volume, Mg.	Color + — per Cent.
O'Donald	10.7	11.5	+7
Swann	12.3	13.1	+6
Redding I	14.0	13.1	−6
Redding II	11.6	10.8	−7
Cheveaux I	10.3	10.7	+3
Cheveaux II	10.2	9.8	−4
Cheveaux III	9.8	11.1	+6
Cheveaux IV	7.3	8.2	+12
Cheveaux V	9.6	9.7	+1
Cheveaux IV	14.0	13.1	−7

Three day periods were obtained in four infants (the infants were fairly well nourished and gained during the experimental period). Various modifications of milk were used. In all of the experiments a negative balance was obtained. The cholesterol content of the stools was from one and a half to three times the amount of cholesterol found in the milk. The quantity of cholesterol in the stools was highest on whole milk with marked constipation. It was

and then two volumes of petroleum ether (distilled below 65 per cent.) Shaking out twice with this quantity of petroleum ether was found to give a complete separation of cholesterol from the digest. By washing the combined extracts once with distilled water the cholesterol was obtained in an entirely clean condition so that after evaporating the petroleum ether and taking up the residue in chloroform a pure green and accurately readable color was obtained when the color developing reagents, sulphuric acid and acetic anhydrid, were added.

We have also applied this method to the determination of cholesterol in blood samples and find that we much prefer it to the method of Bloor because of the regularly dependable quality of the color obtained from residues in this manner.

also quite high during a period with diarrheal stools. This is of especial interest as one might reasonably expect constipation or diarrhea to interfere with absorption of the cholesterol of the food and with the reabsorption of cholesterol excreted into the intestine. The cholesterol in milk is to a large extent associated with the butter fat and its quantity can be changed easily by varying the amount of fat given. There was about one half the amount of cholesterol in fat free milk as in whole milk. The smallest quantity of cholesterol excreted was during the fat free milk period. During all the other periods there was the usual amount of fat present in the food. The composition of the milk as regards salts and protein bore no relation to the excretion of cholesterol.

TABLE 2.—CHOLESTEROL BALANCE EXPERIMENTS—THREE-DAY PERIODS—FIGURES FOR TWENTY-FOUR HOURS

Name of Patient	Food	Cholesterol in Milk, Gm.	Cholesterol in Stools, Gm.	Cholesterol to Body —	Character of Stools	Weight of Stools, Gm.	
						Fresh	Dry
O'Donald*.....	Whole milk mixture 12-20 + 1 oz. lactose 660 c.c.	0.050	0.114	—0.064	Normal	...	6
Swann.....	Whole milk mixture 12-20 + 1 oz. lactose 825 c.c.	0.072	0.212	—0.140	Normal	29	7
Redding I..... 18 mos.	Fat-free milk 1,170 c.c. + lactose	0.056	0.088	—0.032	Formed	30	8
	Whole milk 1,170 c.c.	0.125	0.424	—0.299	Very dry and hard, "soap stools"	42	14
Cheveaux I†	Diluted 16% cream 660 c.c. and lactose	0.075	0.160	—0.085	Soft, thin	27	7
Cheveaux II	Breast milk 620 c.c.	0.107	0.177	—0.070	Soft, thin	...	5
Cheveaux III	16% cream and whey 660 c.c.	0.105	0.242	—0.137	Soft, thin	...	8
Cheveaux IV	Protein milk without whey 660 c.c.	0.044	0.162	—0.118	Formed	24	5
Cheveaux V	Whole milk mixture 12-20 + 1 oz. lactose 825 c.c.	0.071	0.191	—0.120	Diarrheal	217	11
Cheveaux VI	Whole milk mixture 12-20 + 1 oz. lactose 825 c.c.	0.070	0.125	—0.055	Normal	33	6

* Stools and urine collected together.
† Protein in milk, 0.5 per cent.; nitrogen balance for period, 0.31.

We feel that the consistently large negative balance found in these fairly normal and gaining infants while receiving the usual amount of cholesterol in their food is strong evidence in the direction of proving that cholesterol can be readily synthesized. We began these experiments with the preconception that the cholesterol could not be synthesized and had every intention of finding a positive balance. We

proposed then to determine the level of cholesterol intake necessary to maintain a positive balance, and to advertise the dreadful danger of a milk modification inadequate as regards cholesterol. The results obtained have, however, the greater merit that they do not add a complication to the technic of infant feeding.

CERTAIN NUTRITIONAL DISORDERS OF CHILDREN ASSOCIATED WITH A PUTREFACTIVE INTESTINAL FLORA *

LANGLEY PORTER, M.D., G. B. MORRIS, M.D.

AND

K. F. MEYER, M.D.

SAN FRANCISCO

It has long been recognized that the intestinal flora of the very young varies, and that the character of the ingested food is one factor in the production of this variation. The description of acidophilic, gram-positive organisms in the stools of breast fed infants by Escherich [1] and Moro [1] are classic, and the same authors, as well as Bahart,[1] Giarre,[1] Concetti [1] and Schikora,[1] with equal clearness have described bacteria to be found in the stools of artifically fed infants. Thus it has been common knowledge for many years that infants fed a diet rich in bovine milk acquire a characteristic stool flora which in contrast to that of the nursling is putrefactive in its activities and which produces chemical substances (indol, phenol, skatol, etc.) which are known to arise from the splitting of protein. Giarre,[1] Schikora,[1] Langstein,[1] Soldin [1] and Bookman [1] all recognize the antagonism between the acidophilic flora produced under human milk and other high sugar feedings and the alkali loving proteolyzers that inhabit the gut of children taking bovine milk or other foods high in animal protein. Finkelstein used the powers of high casein as contained in his albumin milk to check acid diarrheas brought about by an excessive carbohydrate intake; and for years many of the putrid diarrheas have been treated empirically by the use of cereal decoctions, or of maltose or lactose solutions; the rationality of this procedure is explained if we turn to the results of Kendall [2] and of Torrey,[3] who have shown that under an exclusive carbohydrate regimen,

* From the Department of Pediatrics and the George Williams Hooper Foundation for Medical Research, University of California Medical School, San Francisco, Calif.

1. Quoted by Tobler: "Algemeine Pathologische Physiologie der Ernährung und des Stoffwechsels im Kindesalter." Bibliography, pp. 236 to 247.

2. Kendall, A. L.: J. M. Res. **25**:117, 1911.

3. Torrey, J. C.: J. M. Res. **39**:415 (Jan.) 1919.

putrefactive organisms may disappear from the intestines of experimental animals.

We have long felt that there was clinical reason to suspect an etiologic relationship between protein splitting bacteria and certain types of alimentary intoxications that occur during the earlier years of life. The types of alimentary intoxication in which we have been interested and which we have had an opportunity to study, are:

(a) A mild type with constipation, lack of gain, pallor, head sweating and loss of turgor, the so-called balance disturbance of the Finkelstein school.

(b) A fulminant, acute type with sudden onset, profound cumulative toxemia, tremendous shock, alarming loss of weight and too often an early fatal issue with acidosis.

(c) A grave, chronic type with diarrhea which occurs for the most part between the tenth and twentieth months, characterized by frequent stools, anorexia, progressive loss of weight and increasing intoxication. Previous authors have classed these cases as "Coeliac disease, pancreatic insufficiency and chronic alimentary intoxication."

(d) Certain sporadic, acute, putrid diarrheas of young infants.

(e) A type of chronic malnutrition to be found in older children, a type which in its clinical features corresponds to the balance disturbance of infants, but in which the symptoms are modified by the difference in the ages and dietetic habits of the patients.

The most important and puzzling to explain of these clinical pictures is undoubtedly the acute type of alimentary intoxication, with the dramatic suddenness of its onset, its rapid course, and its tragic termination in death with acidosis. It was this type of case that first fixed our interest in the subject.

Many years ago one of us was struck by the resemblance between the symptoms of intoxication as they occur in this disease and in acute intussusception of infants. Two patients lay side by side in the wards of Lane Hospital, one brought in with an intussusception of some days standing, the other a victim of acute alimentary intoxication. Both were robust, well developed children. Both lay in profound shock, pale, with sunken eyes, flaccid, slightly cyanotic about the mouth, semiconscious; once roused both quickly relapsed into stupor. In the case of the child with intoxication, the symptoms of bowel obstruction failed and the respiratory evidences of acidosis were more marked in type and more rapid in onset. In other respects there was a

startling resemblance in the clinical aspects of the two cases. The similarity was so striking that it emphasized the idea that a similar toxin might be working in the two cases, so that later, when Whipple reported his views on the cause of death in bowel obstruction with the experiments that led him to incriminate a toxic albumose, it was natural to search for a possible source of toxin formation, and with Torrey's classical research on the damaging effects of a high proteolytic flora in the intestines of patients, victims of typhoid infection, it was equally natural to initiate an investigation of the rôle putrefactive organisms might play in acute alimentary intoxications.

In San Francisco the acute fulminating types of intoxication are rare, so that the deductions drawn from the few cases on which this part of the study is based can be only inconclusive, although the facts elicited are striking enough to be suggestive, especially when they are taken with the results obtained from the study of a large number of the more frequent subacute types of intoxication.

The opportunities afforded one of us to study the intestinal bacteria of the three earlier cases were inadequate, but in all the change from the normal flora was striking. The stools of all showed putrefactive organisms in great predominance. All three of these patients died, and in each case characteristic necropsy findings were revealed, great increase in fat in the liver, acute degeneration of the kidney with fatty infiltration, and an almost pure proteolytic flora in the lower ileum.

Shortly after the death of these patients the other collaborators became interested in the problem, and they have developed the bacteriologic and experimental facts here presented.

Following these investigations a system of treatment was devised and in nine cases during the last two years there has been but one fatality, and that within the last month. We feel that the failure in this case was due to the fact that the patient came to us late in the course of the intoxication. The plan calls for lavage of the stomach with a 2 per cent. sodium bicarbonate solution, and the washing out of the bowel with from two to six quarts of a 5 per cent. solution of the same salt, two or three times a day. All food is withdrawn, except that the patient is given large quantities of an iced, 7 per cent. solution of lactose with 2 per cent. of the sodium bicarbonate solution by mouth, and this is continued in spite of the vomiting. We find that the patients suffer less if they are supplied with large quantities of

fluid, and the vomiting ceases more promptly, especially if the ingested fluid is kept very cold.

The intravenous injection of isotonic solution of glucose we have made a routine, even in those cases in which some sugar appears in the urine. If the respiratory signs of acidosis become alarming, Fischer's solution is injected into a vein in a quantity appropriate to the age and weight of the patient. Undoubtedly, the fact that most of these patients were seen before serious acidosis had set in made the treatment more effective, but none the less they were all alarmingly ill and the improvement in each case began at a time when we were able to show that the intestinal flora had altered in type from putrefactive to fermentative.

The following case is illustrative of this type:

CASE 1.—Baby Lane, a patient of Dr. Herbert Yerington, was seen with him at the Lane Hospital in October, 1918. The child was 3 months of age and weighed 13 pounds. It had been on the breast up to September 27 when the mother developed a fever which was diagnosed as typhoid October 2. On this date the child, which had been nursing in spite of the mother's fever, was removed from the breast. It was isolated and put under typhoid precautions. The examination was negative, except that the history states that the child was pale, with dark circles under the eyes, somewhat lethargic and the musculature was soft and flabby and the patient had a toxic appearance. There was nothing striking about the temperature, pulse or respiration. There were several stools which were liquid and had a foul odor. The Widal test was negative and the stool culture showed no typical organisms.

This child was put on 5 ounce feedings of a certified cow's milk mixture of approximately 2-1.75-2.5. From October 2 to 5 the only change was a slight rise in temperature, loss in weight and a fall in the number of white blood cells to 4,000. The stools were still frequent but more solid and very foul. Castor oil was given and the milk reduced and October 7 it was noted that the child's condition was improved. October 8 the condition was about the same, but the child looked more toxic. October 9 protein milk (albumin milk), 20 ounces, water 14 ounces, Dextri-maltose ½ ounce, was given, and the next day the child had twelve liquid stools and eight on the following day with great loss in weight, and depression of the fontanels and anorexia developing.

October 12 the child was about the same and was given a colonic flushing and paregoric and caffein. The formula was changed to a condensed milk in dilution, alternating with a wet nurse breast. October 13 there was no improvement, anorexia was profound, what food was taken was vomited and the stools were very foul. The stool was again searched for typhoid and no colonies were found. However, the examination showed a preponderance of proteus and some streptococci. The same day at 11 p. m. one of us saw him for the first time. The child was very toxic, restless, crying occasionally, throwing the head from side to side, and then relapsing into lethargy. The fontanel was retracted, the lips were dry, there was an extreme pallor, the

temperature was 100 F., the respiration was irregular and the child had the appearance of impending death.

It is interesting to note the onset and rapid culmination of these more profound symptoms after the use of protein milk. The child was immediately ordered a massive lavage of the bowel with alkaline solution, a Murphy drip of 5 per cent. glucose, the subcutaneous injection of physiologic solution of sodium chlorid and by mouth a 10 per cent. solution of Dextri-maltose in water, 3 ounces every two and a half hours, and frequent small doses of water by mouth from a medicine dropper. It is noted on the history that October 14 the general condition was greatly improved, the toxemia diminished and that the bowel movements had become fewer in number and less foul. Small doses of brandy and paregoric which had been given before were continued, alternating every four hours. A small amount of breast milk was added to the maltose mixture, but was withdrawn the next day.

October 20 the toxemia had entirely disappeared, the child's stools were two in number, soft, yellow and without mucus. October 24 the child was able to tolerate a mixture containing one-third milk, but with a very high percentage of carbohydrate. December 2, when the child was returned to a normal formula, the picture was that of a healthy, happy baby gaining an ounce a day. Coincident with this reestablishment of well being, there was an entire displacement of the putrefactive organisms present during the height of the illness. Such a case represents the less grave form of acute alimentary intoxication. Cases with fulminant onset which run a more rapid course, depart from this picture only in degree.

However, there is still another clinical entity which should be classed as an alimentary toxemia, in which the progress of similar symptoms is slow, and the onset insidious. In such patients the development of a profound toxemia runs through a period of weeks, sometimes months, until it finally merges into a state of chronic malnutrition, or ends in death from starvation. In cases of this type the most striking symptoms are anorexia, occasional vomiting, and a mild diarrhea in which the stools are pale, usually foul, sometimes with a curious mousy odor, rarely odorless. Very often a large quantity of liquid is evacuated with a smaller mass of fecal material imperfectly mixed with mucus; in the severest cases the mucus may contain more or less blood and pus, and in the later stages of the attack undigested food appears in large quantity; at first fat is not wholly split; as time goes on, unaltered fat appears in greater quantities, and later unhydrolized starch and meat fiber also are to be found. Naturally, with such a failure of digestion, weight is lost rapidly while earthly pallor, sweating and the other facial indexes of intoxication appear. A few of these patients develop an intensely itching erythema of the skin, and together with this a curious lethargy with what might be called a pseudocatatonia. These children assume the most extraordinary postures and

maintain them for hours at a time. Necropsy on such a patient revealed no abnormality other than moderately large mesenteric lymph glands, fatty liver, and a preponderance of putrefactive flora in the ileum.

An example of this more usual type of chronic alimentary intoxication is:

CASE 2.—G. S., 17 months of age, had an uneventful history until the tenth month when he was weaned from the breast and given a feeding of equal parts of top milk (about 10 per cent. fat), and water with two teaspoonfuls of lactose to each ounce feeding, which was given every three hours. He did well until March 1, 1919, when he developed vomiting and diarrhea. Six to eight very offensive stools were passed daily together with much gas. Reduction and dilution of milk and the prescribing of castor oil produced no amelioration. The child was depressed, irritable and "very sick."

He continued to fail while the symptoms increased in intensity until April 8 when one of us saw him for the first time. He was then very toxic, he vomited ingesta, even water. He alternated between restlessness and lethargy. His breath bore a distinct odor of acetone and his temperature ranged around 103 F. He showed an extreme loss of fluid and a lack of turgor that was striking. The eyes were sunken and ringed with dark circles, otherwise no pathologic changes could be found by the methods of physical examination. There was a marked degree of muscular hypotonia, the child assumed the most unusual attitudes and maintained them for hours—a condition that might be denominated pseudo-catatonia.

Laboratory findings were negative except for a high degree of acidity in the urine, no acetone, and a predominantly putrefactive flora in the stool.

For two days the child's intake was limited to fluid and lactose, 2½ ounces a day given in the form of "high calory" orangeade. Three hundred c.c. physiologic solution of sodium chlorid was given by the intraperitoneal route on each of three occasions during this time. Six quarts enteroclyses of 1 per cent. of aluminum acetate were used daily, and on alternate days 2 drams of castor oil with 5 drops of turpentine were given. The diet was made more liberal, other carbohydrates were added, and an egg yolk was given once daily. No milk. The patient went on for about three weeks; the afternoon temperature rose to 101 and 102 F. and an indurative erythema appeared on the skin. This itched intensely but responded to the application of compresses of 1 per cent. phenol in glycerin and water.

After three weeks of this treatment, during which the toxemia subsided, the vomiting stopped and the bowels ceased to evacuate abnormally; then came a sudden attack of diarrhea. The stools during this attack showed a high putrefactive quotient. Castor oil and sodium bicarbonate lavages checked the looseness of the bowels. After one day's starvation, one-half ounce more lactose daily was added to the diet. In a few days, cereals, Italian pastes, fruit sauces and jellies were added. Ten days later a stool examination showed a very slight putrefactive flora. The clinical signs were improving daily and the child left the hospital in good condition—a normal, convalescing baby.

In childhood there are other more numerous cases which present various clinical aspects; these may be classed as subacute or chronic alimentary intoxication. In certain of them the clinical picture is dominated by a diarrhea whose chief distinction is its exceedngly fetid odor. Mild, evanescent and recurrent forms are not infrequently found among infants during the second and third half years of their life. Such cases are much more numerous than is generally supposed, and the reputation of many proprietary foods as valuable in diarrhea depends on the fact that high carbohydrate feeding, especially dextrin feeding, is effective in checking them. The butter-mehl soup of Ochenius is also effective for the same reason.

Clinicians have always found such cases, especially the severer ones, exceedingly difficult to treat, but since we have been able to submit the stools to the tests for putrefactive bacteria, and have found that in all of them some one or other of the putrefactors occurs in great predominance, treatment has become more effective. In the very severe cases practically no other than putrefactive organisms inhabit the gut. Acting on the information derived, we have instituted high carbohydrate, mainly lactose, feeding, and at the same time have withheld or limited the animal protein intake. And almost without exception we have had relatively rapid recovery with no recurrence. The following case is illustrative.

CASE 3.—V. S., 14 months of age, referred by Dr. Shade, Oakland, Calif., was breast fed till 11 months of age. Prior history was uneventful. The child had not gained since removal from the breast three months before. She was on a usual diet for her age, including cereals, broth, soup, a little vegetable puree and much milk. Blood and urine were not strikingly abnormal. The stools were typically proteolytic to cultural tests. She had an intermittent fever. The breath bore an acetone odor. There was persistent loss of weight, increasing pallor and anemia, lack of turgor, and lethargy alternating with periods of irritability.

She was given a high carbohydrate diet, but as this was one of our earlier patients and we had not yet come to a full realization of the part milk protein plays in these cases, she was allowed 20 ounces of milk in her formula. Gray powders and Bulgarian cultures were also given. Two weeks of treatment along these lines followed. The vomiting became more persistent, the stools increased to from twelve to fifteen daily; they were very foul and great quantities of fluid were passed with them. Anorexia became positive and weight was being lost in an alarming manner so that gavage was resorted to of necessity. At this time all animal protein was withdrawn, a 2 per cent. sodium bicarbonate solution with brandy by mouth was the only ingesta allowed for twelve hours. Then for another twenty-four hours only this with the addition of iced, 5 per cent. lactose solution was given. Sodium bicarbonate enemas were also given.

The child began to mend slowly. The lactose was now given in the form of orangeade, 3 ounces of the sugar each twenty-four hours. Cereal decoctions, dextrins and other carbohydrates were added. Little by little the symptoms disappeared and at the same time the stool flora showed an increasing movement toward fermentative types. It was more than sixty days before this child was really free from the toxic symptoms and about four months before it resumed a satisfactory state of nutrition. Since then, however, the child has done remarkably well and is fat, healthy and happy.

Still another group of malnutrition cases with symptoms of mild intoxication occurs in young infants exclusively bottle fed, who are constipated, fail to gain weight, are pale and sweat about the head, and exhibit a loss of turgor. These children would be classed by Finkelstein as babies suffering from balance disturbance. Their stools always contain a much higher proportion of protein splitters than do the evacuations of more perfectly nourished babies. Such infants show rapid improvement with diminution of symptoms when they are put on a higher carbohydrate feeding, provided that the sugar in the food is increased gradually.[4] The success of malt soup feeding is most striking with this type of malnutrition, and the result depends on the replacement of a proteolytic by a predominant saccharolytic intestinal flora, and we have many case records to illustrate the rapid improvement of such patients when they are given an appropriate diet.

Among older children there are often encountered individuals whose symptoms suggest the activities of a toxic agent. A proportion of the children classed as neurotic and delicate present this aspect. On close examination their symptoms coincide in a striking manner with those of younger children who are suffering from the chronic types of alimentary intoxication just related. These patients are usually of the school age and they are brought to the physician because of lack of energy, sometimes because of irritability and naughtiness, at other times for lassitude and malaise. Irritability and bad behavior are often indexes of fatigue rather than of excessive spirits. On physical examination the children are usually underweight, and there may have been no gain for months, or even years. Their appetites are capricious, most often they suffer from constipation often alternating with diarrhea. Their color is invariably poor, although the hemoglobin percentage is not always low; there seems to be a toxic vasomotor disturbance which produces this pallor; but the most striking thing about the appearance of such children is the sunken, dark

4. Porter, L., and Dunn, C. H.: Am. J. Dis. Child. **10**:77 (Aug.) 1915.

circled eye. In our experience these little ones have always shown a predominant proteolytic flora, and in every case they have responded to a regimen which included rest in bed with a high carbohydrate feeding. In all instances such patients have remained well so long as the flora in the intestines showed a reasonable proportion of aciduric organisms, and they have relapsed whenever they were returned to a high milk feeding. Such an instance is:

Case 4.—J. A., first came under observation with a cystitis when two years of age. She was not seen again until she was 4 years of age when she was brought in because of an intermittent fever. The child has always been constipated and has slept restlessly and failed to gain in weight. Physical examination revealed a child normal except for dark circles about the eyes, a tired look and small, infected tonsils. Stool examination showed a flora predominantly putrefactive.

The child was put to bed and she was given gray powder, ½ grain, three times a day, and she was put on a very high calory diet, consisting exclusively of starches and sugars. This treatment was followed for three weeks without any change in constipation, but the child's physical condition was remarkably improved, she slept better, but no gain in weight was made. After five weeks, sweets, vegetables and fats were added. After a further three weeks the stools showed only a mild proteolysis but the child's constipation was not any better. Acidophilus cultures were given and the constipation began to improve and the stool was reported to be completely fermentative. After another six weeks the girl was put on a diet in which carbohydrates preponderated, and the acidophilus cultures were continued. The constipation had entirely disappeared and there has been no recurrence and her general health is perfect. The child has steadily gained weight.

One other consideration may be discussed. Torrey's[5] original paper was written to demonstrate the evil effect of the high putrefactive flora as a complication in typhoid fever, and from his plea came the utilization of high carbohydrate feeding in that disease. Undoubtedly, in many other diseases, especially during infancy and childhood, the handicap of a putrefactive intestinal flora tends to complicate the clinical picture and to make the condition graver. This is especially true in cases of sepsis in infancy, and undoubtedly in all the acute infections, especially in certain cases of pneumonia and in the bacillary dysenteries of infants, great advantage will accrue from a study of the flora and a substitution of high carbohydrate feeding in place of the excessive quantity of milk that is often fed to these patients. A case of sepsis so complicated which undoubtedly would have proved fatal without a recognition of these truths was the following case:

5. Torrey, J. C.: J. Infect. Dis. **16**:72 (Jan.) 1915.

CASE 5.—E. G., was referred by Dr. O. Stansbury, Chico, Calif. The family and past history was entirely unimportant up to the onset of the present disturbance. The complaint was that five weeks before she was first seen by us the baby had had what was termed a severe bronchitis with a temperature of 102 F. She was very restless, deeply lethargic and she was continually bathed in perspiration. Her pallor was alarming and she cried as if in pain. She was slightly constipated. There was no diarrhea at any time. The cough gradually improved and the baby became somewhat better. Ten days previous to the day on which she was admitted to the hospital the child had refused to nurse and she then had a slight fever. She again became decidedly lethargic and developed at this time a high grade of photophobia, due to a conjunctivitis. Her turgor was poor because of the great loss of weight evident.

On physical examination we found a rather restless child moaning in her sleep, rolling her head and keeping her eyes continually closed. The conjunctivae were very much reddened. The central nervous system was normal. The eyegrounds were perfectly normal. The temperature was continuously 101 F. in the afternoon. The blood count showed a hemoglobin percentage of 85, with normal white cell count and differential ratio. The urine showed a slight trace of albumin. The stool flora was predominantly putrefactive. The von Pirquet was negative. Streptococci were found in the throat culture. The conjunctivae were not cultured nor was the blood, but clinically this was a typical picture of a streptococcic infection of the blood stream with localization in the upper respiratory tract and the conjunctivae. The case was complicated, however, by the presence of the highly putrefactive flora which we found in the stools.

The child was put on a diet of 7 per cent. lactose solution, alternated with 1.5 per cent. sodium bicarbonate solution, 3 ounces every two hours. This feeding was continued for two days, then 3 per cent. cereal water was added to 7 per cent. lactose, and 0.4 per cent. of protein was allowed. Carbohydrates were given in quantity, fats in moderation, while the protein in the mixture was increased slowly.

The symptoms of the self-limited blood stream infection passed away in about two weeks and the mother was allowed to leave the hospital. She tried to increase the milk in the food to an amount appropriate for a healthy child of this age. Immediately there was a relapse and a return of the symptoms of intoxication. The milk was promptly withdrawn, lactose cereal decoctions, fruit juices and dextrins were given. After one month the turgor was good and the child had regained much of its weight. Its stools were normal, it had lost all its lethargy, had become playful and happy, and at the same time an examination of the stool showed a return to a balanced flora.

While we have been studying the clinical aspects of the various alimentary intoxications for some time, the more intensive bacterial studies are the work of the past year and one half. A detailed account of the bacteriologic methods worked out for the investigation of the problem will appear elsewhere.[6] In this communication we can do no more than indicate the principles on which we have proceeded, and

6. J. Infect. Dis. (in press).

Abbreviated Form of Stool Analysis

Culture Medium	Normal Stool			Putrefactive Stool			Fermentative Stool		
	24 Hrs.	48 Hrs.	72 Hrs.	24 Hrs.	48 Hrs.	72 Hrs.	24 Hrs.	48 Hrs.	72 Hrs.
Gelatin tube	Liquefaction +	Liquefaction +	Liquefaction ++	Liquefaction ++	Liquefaction +++	Liquefaction +++	Liquefaction —	Liquefaction —	Liquefaction +
Loeffler's serum slant	Digestion and discoloration +	Digestion and discoloration +	Digestion and discoloration ++	Digestion and discoloration +	Digestion and discoloration +++	Digestion and discoloration ++++	Digestion and discoloration —	Digestion and discoloration —	Digestion and discoloration +
Cresol-purple milk tube	Acid reaction Gas + Whey expression + Clot soft	Peptonization +	Peptonization +	Acid reaction Gas ± Whey expression + Clot soft	Peptonization ++	Peptonization ++	Acid reaction Gas ++ Whey expression +++ Clot firm	Peptonization —	Peptonization —
1% lactose peptone bile	Gas production 15 to 30 per cent. Smear of sediment; B. coli, gram-positive cocci; few gram-positive rods			Gas production 10 to 25 per cent. Smear of sediment; B. coli, gram-positive cocci; few gram-positive rods			Gas production 40 to 50 per cent. Smear of sediment; B. coli, few aciduric		
Endo plate	Reaction, acid Growth ++ Odor, acid Type of colonies, varied			Reaction, alkaline Growth +++ Odor, pungent and offensive Type of colonies, numerous			Reaction, acid Growth + Odor, strongly acid Type of colonies, few		

describe a technic which we believe to be accurate and at the same time simple enough for clinical application.

No reference will be made here to the qualitative side of the work which consumed many months and from the results of which we were able to take a few qualitative tests that have been combined to form a clinical diagnostic technic. For this purpose it seems four tubes and an Endo plate are amply sufficient. The tubes are two solid ones—one a Loeffler's serum, the other a gelatin stab—and two fluid tubes, the first a 1 per cent. lactose peptone bile, the second a cresol purple milk. The most striking change happens in the latter when such a tube is infected from a predominantly fermentative stool; there is great retraction of the clot, and a maximum expression of whey. After planting from the putrefactive stool the resulting clot is soft, there are few or no gas bubbles, and a trifling amount of whey is forced out; frequently there is stormy fermentation. Seedings from normal stools give appearances in the tube that more nearly approach those of the putrefactive tube than of the fermentative, although they are in themselves distinctive.

The lactose peptone bile gives most aid when the gram smears of the sediment are studied. Here we find the representation of the viable flora of the stool. In proteolytic stools the picture is dominated by spore bearers, gram-positive cocci and *B. coli*. When the gas in this tube exceeds 60 per cent. we can be sure that *B. welchii* predominates in the stool.

When smeared from a putrefactive stool, Loeffler's serum tubes after seventy-two hours show marked digestion, which has been recognizable from the twenty-fourth hour. They present a characteristic dark brown discoloration. The odor is also pathognomonic. It is strong and very offensive. Fermentators growing on this medium cause no digestion, produce no odor and thrive poorly.

Gelatin stabs from fermentative stools have remained unchanged, while those made from putrefactive evacuations are completely liquefied. Liquefaction begins during the first day and is complete by seventy-two hours. The Endo plates give such striking contrasts when streaked from proteolytic and saccharolytic stools that we feel that they will be a great aid in routine diagnosis. The putrefactors produce a strong, characteristic mousy, musty odor from an abundant growth, which causes an alkaline reaction to the medium, thus leaving it unchanged in color. While, on the other hand, fermentative organ-

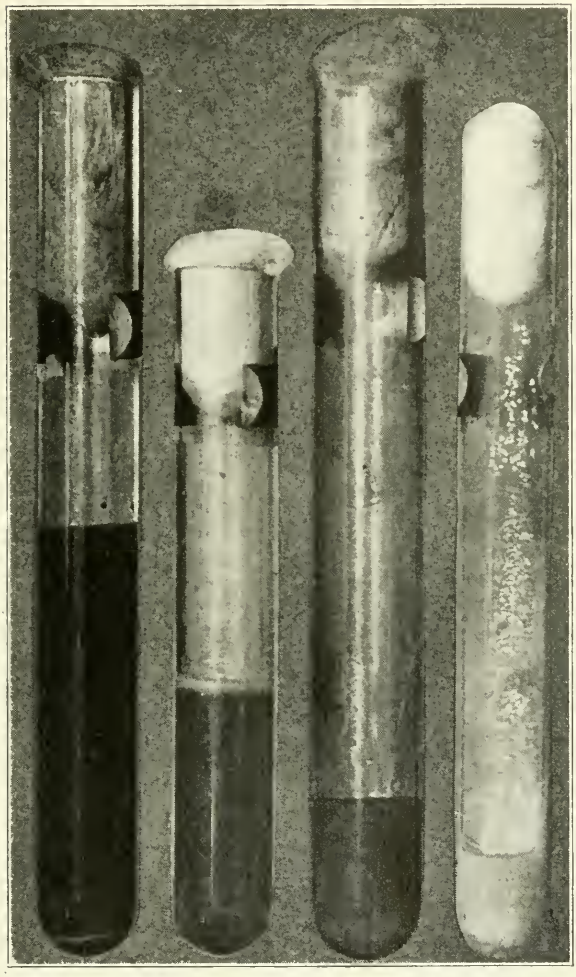
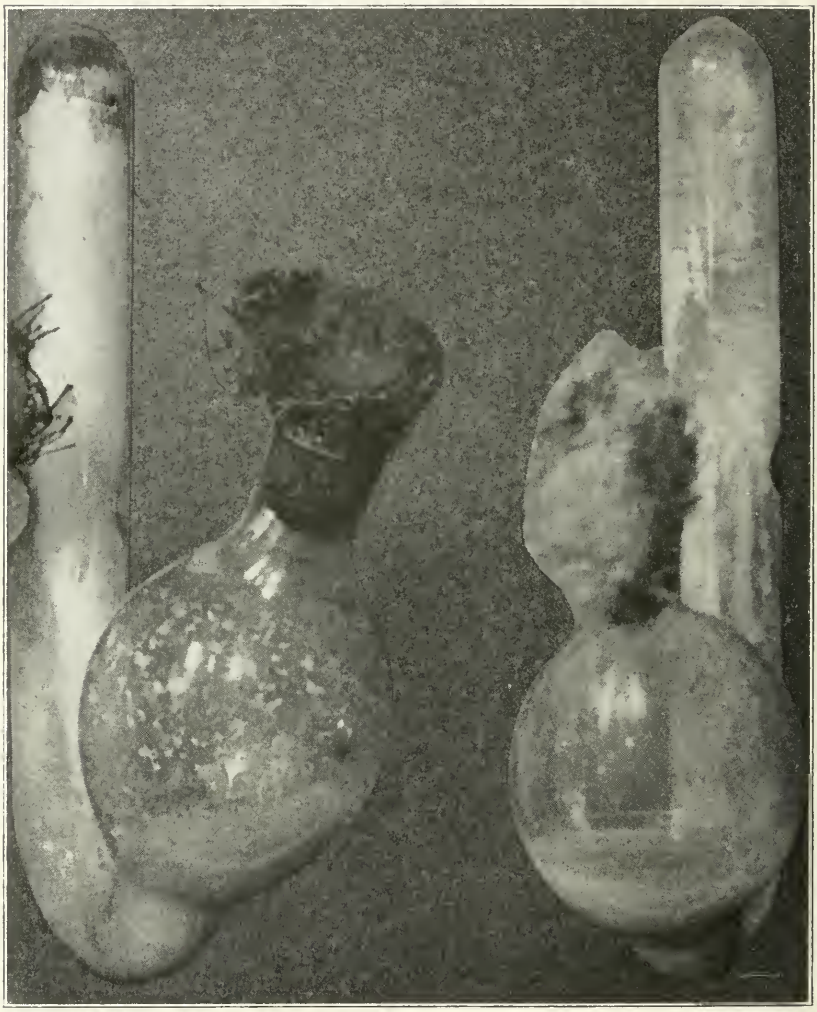
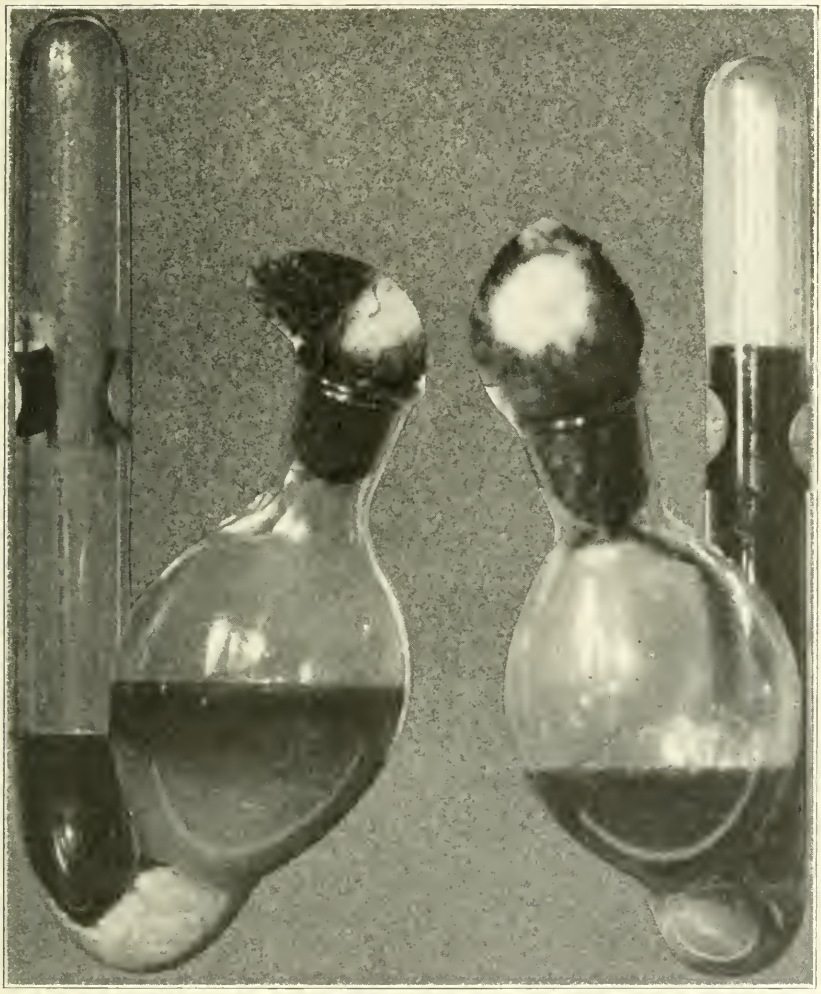


Fig. 1.—A 1: Gelatin tube inoculated with putrefactive stool specimen. Complete liquefaction and uniform turbidity. A 2: Gelatin tube inoculated with a fermentative stool specimen. No liquefaction and surface growth. B 1: Loeffler's serum tube, complete proteolysis of serum produced by bacteria of a putrefactive stool. B 2: Loeffler's serum tube, absence of proteolysis—fermentative stool.



A B

Fig. 2.—A: Cresol purple milk—result of a putrefactive stool specimen. B: Cresol purple milk—result of fermentative stool specimen. Note the expression of whey.



A B

Fig. 3.—A: Lactose peptone bile—gas production when inoculated with fermentative stool specimen. B. Lactose peptone bile—gas production when inoculated with a putrefactive stool specimen.

isms on this medium grow scantily and give a strong odor of acetic acid, and because of this acidity produced a color change from the normal pink to a deep red.

These simple tests, which can be done in any office laboratory, enable us to verify our clinical suspicions quickly and with certainty.

We have been enlightened and encouraged by the experimental data which Torrey[7] has recently supplied. His studies amplify and reinforce the conclusions that we have been able to draw from our own experiences. His data, fully presented, enable him to state as a fact that in dogs the intestinal flora responds promptly and with uniformity to dietetic changes. In his dogs he was able to produce an acidophilic flora at will by feeding lactose and dextrins. He states that one would expect to find a more clearly marked response to food stimuli in the flora of stools taken from the comparatively short canine colon than in those evacuated from the longer human intestine which favors stasis. Torrey further points out the fact that while *B. welchii* is functionally fermentative, it is found always associated with large numbers of putrefactors—a finding which, perhaps, explains why the apparently paradoxic method that calls for two days pure protein diet followed by high sugar feedings is successful in eliminating an excess of Welchii from the stools as well as in removing the toxemias produced by the concomitant putrefactors.

After a study of the facts so far derived from their observations, we feel that we have been able to show (1) that children whose diet is well balanced and whose nutrition is normal, have an intestinal flora consisting of fermentative and putrefactive types without preponderance of either; (2) that children fed on large quantities of cow's milk have a more complex flora made up of various types, most of which are facultative putrefactors; (3) that in children who suffer from certain of the types of alimentary intoxication with malnutrition, the intestinal flora departs in a uniform manner from the normal, and that this departure is always characterized by the establishement of bacterial types predominantly putrefactive; (4) that the return of these children to normal health is coincident with a regression of the intestinal flora toward predominantly fermentative types and a later swing to balance between the two types; (5) that such changes in the intestinal flora can be brought about in the intestine of the human infant by withdrawing animal protein and persistently feeding large

7. Torrey, J. C.: J. M. Res. **39**:415 (Jan.) 1919.

amounts of lactose (from 2 to 4 ounces daily), and other carbohydrates; that the period which may be necessary to produce this variation is from ten to forty days. While feeding acidophilus cultures has in a few cases aided a more rapid establishment of aciduric flora in the baby's intestine, this influence was not very great; (6) that the progressive cessation of the symptoms of intoxication and a return of toxemic patients to nutritional health coincides with the recognizable dominance of a fermentative flora; (7) that lactose and dextrins are the carbohydrates most effective in encouraging the rapid establishment of a fermentative flora in the intestines of infants and children.

240 Stockton Street—Second and Parnassus Avenues.

DISCUSSION

DR. KOPLIK: I wish to call attention to the divergence of opinion between Czerny and Finkelstein. The Czerny school insists on high carbohydrate and low fats, and the Finkelstein advocates low fats and high protein. I do not wish to criticize the milk Dr. Porter used, but a great deal depends on how protein milk is made. Finkelstein had his own plant for making this milk put in. Unless protein milk is made properly I think it rather hurts the baby and may cause a bloody diarrhea. Protein milk is very difficult to manage. The milk may be stored too long after it is prepared and here we are in the hands of the purveyors of milk. In such cases as Dr. Porter describes I have been accustomed to cut out the sugar. Dr. Porter gives lemonade while I cut out all sour foods.

TRANSFUSION IN INFANCY AND CHILDHOOD—INDICATIONS AND DANGERS

HENRY KOPLIK, M.D.

NEW YORK

There is nothing novel in transfusion, for we are told (Crile) the Egyptians practiced this method of infusing new life into human beings. Their methods were necessarily crude. It is not the purpose of this paper to trace the developments of these methods into the refined procedures of the present day. It is enough to say that with the impetus given by experimental work, the whole realm of transfusion, and even the use of human blood serums, has been vastly improved; so that today we have at our command a method of blood therapy which is capable of actual life-saving effects. The procedure varies with different operators, but it all crystalizes itself, I imagine, in the skill of the modern surgeon to handle blood vessels in the transfer of blood from one human being to another in such a manner as not to endanger life. Formerly transfusion was accompanied by dangers, one of which was the admission of air into the circulation. These dangers today are not only avoidable, but if they occur are inexcusable.

In the field of transfusion in new-born infants and children vast advances have been made, and this in the direction of life saving, which are unmistakable. In the new-born, hemorrhage, either as a sign of diathesis or sepsis, is successfully combated by the introduction of serum or blood if properly performed. The history of melena, it is true, before the days of serum injections or transfusion, was not a hopeless one. I remember well a severe case of melena in an infant 4 days of age, in which the amount of blood lost was very great, so that the infant became exsanguinated, yet recovered quite well with the subcutaneous introduction of gelatin. I would scarcely recommend this method today, inasmuch as it is not so uniformly successful as other more easily applied methods. The introduction of gelatin is not without danger; if incompletely Tyndalized tetanus is apt to supervene upon the hemorrhagic phenomena.

I would classify the application of transfusion in the new-born as follows: (1) in cases of uncontrolled hemorrhage from wounds, umbilicus, or scalp hematoma: (2) in forms of frank melena with

hemorrhage from the intestines, the subcutaneous hemorrhage and gastric hemorrhages; (3) concealed melena in which the outward objective signs of hemorrhage are slight, such as an occasional ecchymosis on the surface, but in which the evidence of internal hemorrhage is great, such as critical loss of weight, intense anemia amounting to exsanguination, very slight signs of hemorrhage from the intestine, and with marked hemorrhage into cavities such as the subarachnoid space. (4) There are cases of severe and continued hemorrhage in the new-born from surgical wounds such as circumcision, hemorrhages which are of a parenchymatous character and are exsanguinating to a degree. In these cases we must conclude, as in the cases in which the hemorrhages occur beneath the scalp, that there is a congenital condition present, a hemophilia.

In hemorrhage of the new-born, the greatest success occurs in cases in which the condition is promptly met by transfusion, and this in a positive manner. I mean that former procedures have contented themselves with the introduction of either placental serum or quantities of normal serum, with great measure of success. From the failures which I have met with these methods, I would say that the greatest success lies rather in the direct introduction of blood into the circulation of the patient. In one stroke the patient is saved the anxious waiting and reintroduction of serum in case of failure to control bleeding.

There are two methods now before us to choose from, and the time is scarcely ripe to decide positively in favor of either. The one method is that of the direct introduction of full blood from a donor to the patient's circulation, the Unger or the syringe method (Lindemann); and the so-called citrated blood method (Lewisohn). The advantage of the direct method with blood unadulterated is evident if in all cases we can test the donor and recipient for hemolysis. The advantages claimed for the method of citrated blood are, first, that we can transport the blood from donor to recipient at some distance; again that in a great emergency the tests for hemolysis are not necessary in the citrate method. There is some discussion, that in the citrated method repeated transfusions are apt to act in a hemolytic manner; that citrated blood often used may accomplish the hemolysis which we endeavor to avoid and thus favor hemorrhage. In my own experience, I must confess, I have no positive evidence clinically that such a change occurs. I have made use of both methods of transfusion, and in the new-born both methods are applicable; that is, the direct transfusion

of Unger and the indirect of Lewisohn have both been successful. However, this may be chance and individual skill in technic.

The Unger method has the appearance of immediate success, and so the citrate method by the *author* of the method. In many cases the surgeon still lacks the delicate skill in technic and many failures may be due to this, and it is extremely dangerous to conclude that any one method is superior to another unless all cases came out of the hands of the same operator who has handled both methods. After all, unadulterated blood introduced direct into the circulation is preferable to any blood which contains a substance foreign to it. At the same time an unskilful operator may fail with any method, so that with improved methods the unadulterated blood will hold its own as a transfusion agent, if the surgeon will show the necessary delicacy in technic. I have never seen a failure which I could attribute to the use of full normal blood or citrated blood. It has always been the technic which I could criticize. Some surgeons are unable to find the proper vessel, hence the uncovering of other methods, such as the use of the larger veins and sinuses (longitudinal). These latter-day methods (jugular vein and longitudinal sinus) to my mind show an intensive seeking of an easy avenue of introduction of the blood into the body.

In the new-born it is rarely necessary to repeat a transfusion and it is for this reason that I have always insisted upon transfusion from the start. I have seen cases in which the subcutaneous introduction of serum, or even the subcutaneous introduction of whole blood, has absolutely failed to control hemorrhage. In these cases, if the patient lived, a single definite transfusion succeeded in combating symptoms which the serum and subcutaneous introduction of blood failed to control. The improvement in the patient's condition took place even where symptoms of sclerema, which is often regarded as a forerunner of a fatal issue, set in. Temperature also is not a contraindication to transfusion. In one case a transfusion was done by the Lewisohn method there was a cephalohematoma extending over the whole scalp down the neck. One transfusion of 100 c.c. of citrated blood controlled the hemorrhage completely. In this case there was sclerema of both lower extremities and beginning sclerema of the upper extremities, ecchymotic spots on the elbow and ankle, edema of the face, blood on the umbilical stump, but no bleeding. The case was one of melena in a hemophilic. It may be of interest

that the coagulation time (slide method) was twelve minutes; hemoglobin 24 per cent., white blood cells 38,000, red blood cells 1,800,000, and still a recovery took place. This patient was discharged apparently cured and was readmitted a week after discharge from the hospital. This time the red blood cell count was 4,200,000; the hemoglobin 43 per cent., coagulation time eight and a half minutes. The infant was transfused again by the Unger method, but the patient did not recover. It was in this case that an injury and neglect of the scalp, started the hemorrhage again. On no other ground can the recrudescence be explained, as the infant was increasing in weight when discharged from the hospital after the first transfusion. The infant upon readmission to the hospital showed evidences of extreme neglect.

Congenital syphilis with melena does not contraindicate transfusion, as seen in one case. The infant, having been in extremely poor condition, made a good recovery with 80 c.c. of the father's blood. The great dangers in transfusing the new-born are, first, the introduction of too great a quantity of blood, full or citrated. Some operators seem to be guided by a desire to give as much blood in quantity as they can, and the result is disastrous. I have rarely found that more than 80 c.c. or 100 c.c. were necessary. If the amount can be estimated or if the hemoglobin is calculated, it need not be increased more than ten points. The recuperative powers of the body will accomplish greater recovery after the transfusion. In new-born and older infants a very great danger is too rapid transfusion. In those cases in which there was haste in performing the operation, the patient did not, at least in my experience, do well no matter how much or how little blood was transfused. The recipient developed edema of the lung due to heart weakness. I surmise sudden overloading of a weakened circulation.

In the new-born, though in some emergencies the subcutaneous introduction of blood does well, many patients who are lost by simple subcutaneous infusion of blood or serum might have been saved by the more radical method of transfusion. An infant, 7 weeks old, was bleeding from a circumcision wound.. In this case two injections, 20 and 35 c.c. of whole blood, were introduced into the tissues of the buttocks of the infant without success; a 100 c.c. by direct transfusion (Unger method) caused a cessation of bleeding from the wound.

TRANSFUSION IN LATE INFANCY AND CHILDHOOD

Hemophilia.—In hemophilia transfusion is uniformly successful in combating the hemorrhages even though large amounts of blood have been lost. In one case the hemoglobin was reduced to 21 per cent. and the red blood cells to 800,000. In such cases repeated transfusion with larger amounts of blood, 200 c.c., became necessary. In one case both the Unger method and the citrated method were employed. Both methods showed the same efficiency in stopping bleeding, in fact the recrudescence of bleeding necessitated repeated transfusion. In these cases also there is a record of a case in which serum in large quantities was introduced subcutaneously in an infant 11½ months old, but without any result. In this case after transfusion the hematoma, the result of an injury, as also bleeding elsewhere, was controlled.

Pernicious Anemia.—This is such a very rare condition in infancy that its occurrence would not be recorded here if I did not have in the examination of the blood and its study the cooperation of Dr. Ottenberg, who, I wish particularly to note, gave much study to the case. I may say that the patient was transfused twice, 140 and 120 c.c. of citrated blood being used. The hemoglobin, reduced as low as 15 per cent. at one time, mounted to 44 and 34 after the transfusion, respectively. The spleen was enlarged and the question of its removal was debated and rejected after frequent consultation. When discharged from the hospital, after six weeks' stay, she certainly was not well, though she was improved. Open air existence day and night seemed to do much for the patient, who today is a handsome child of five years, normal in every way and strong.

ACUTE DISEASES WITH TENDENCY TO HEMORRHAGE

Hemorrhagic Diathesis.—In one case several bleedings both subcutaneous and from the nose were complicated by an otitis, from both of which the patient recovered. The otitis was a complication after admission of the patient to the hospital. This patient showed a hemoglobin of only 18 per cent. at one time.

Leukemia.—Acute and chronic leukemia are not permanently benefited by transfusion. Transfusion in these cases should not be performed as a cure but as an aid to the performance of other therapeutic procedures which may be thought proper.

Purpuras of All Varieties.—These are not improved as to the purpuric condition by the introduction of new blood into the system. One form of purpura is particularly rebellious to treatment. This is Henoch's purpura. I have repeatedly transfused in these cases but have succeeded in only temporarily improving the general condition, such as the anemia, but cannot say that the recrudescence of attacks of pain or hemorrhages with purpura have in any way been controlled.

Sepsis and Acute Infections.—I now wish to discuss transfusion in a series of conditions which are subacute and infectious. In the condition of chronic subacute infectious endocarditis there is a constant condition of anemia, a hemolysis. It would seem that transfusion would lend an aid in bridging over the crises which occur in these cases. In patients who run a low temperature with endocarditic crises due to an infectious endocarditis, of whom I have had several, I transfused in the hope that I might improve the resistance of the patient. I have not had any success. On the contrary, a few days after transfusion, the hemoglobin index, though improved immediately after the introduction of blood, rapidly fell to the initial index, and the patient sank even more rapidly than one would expect even if transfusion had not been practiced.

There is one form of streptococcus invasion in which if transfusion is done intensively and repeatedly, improvement may take place after the introduction of new blood. This is the form of blood sepsis which does not affect the heart itself. In one case of sinus thrombosis resulting from ear infection, as many as eight transfusions tided the patient over repeated crises. The transfusions were performed by the Lindeman method and small quantities, not exceeding 250 c.c., were introduced. The patient did well in spite of complications in the pleura and joints, beginning with thrombosis in the jugular, which could be felt before and seen after ligation of the jugular. In this case there was a typical sinus thrombosis, resulting from chronic otitis in a boy 10 years old. The jugular could be felt thrombosed and by operation only part of the thrombosis could be removed. The thrombus extended as far as the subclavian vein. After the sinus operation the boy developed pneumonia and osteomyelitis. He was transfused by the Lindeman method five times, with blood from a donor who had been vaccinated with streptococci cultivated from the patient's blood. As much as 2,500 c.c. were introduced into the system of the patient at various intervals. The patient finally made a good recovery with simply resultant stiffness in one hip.

In explanation of the method of transfusion in this case, I advised the removal of a certain amount of blood before the introduction of the new blood, and this in my mind was the kernel of success of the treatment in this case. The boy is now well and tramping in the woods. Dr. Finley Bell allowed me to see this patient in consultation a number of times. To my mind the case illustrates in many ways the advances we have accomplished in the therapy of septic infections. In this and similar cases I have advised the removal of blood in order to make way for the reintroduction of new blood in large quantity, thus avoiding the danger of overloading the circulation — bleeding first and then transfusion.

INFLUENZA WITH INVASION OF THE CIRCULATION BY HEMOLYTIC BACTERIA

In the recent epidemic of influenza there occurred cases in which the hemolytic effects of the invasion of the system by the influenza bacteria was quite marked. In one case there was very little involvement of the lung, but the kidney bore the brunt of the infection. In another case, a boy, 6 years of age, had an influenzal nephritis. The urine was the color of Burgundy wine and the sediment of the twelve-hour urine was fully one-eighth of the fluid content; the temperature ranged from 104 to 105; the urine showed a few renal elements or even blood and it seemed as if the urine was colored with the blood coloring matter. The patient had no subcutaneous hemorrhages, but the skin was of an ashy yellow hue, and the patient was fast sinking. A transfusion of whole blood in this case caused not only a cessation of bleeding, but the nephritis disappeared completely. I had occasion to see the patient a few months after transfusion; he had gained 10 or more pounds and was the picture of robust health; there was no trace of kidney lesion. The patient came to me for an asthmatic condition from which he suffered. The saving of the life of this patient could be directly laid to the door of the single transfusion. On the other hand, many patients with the same affection are transfused without success; the physician waits too long. I saw a similar case after influenza in which the patient had a severe hemorrhagic nephritis. He had lost an amount of blood, when an acidosis or acetonemia set in. In this case the transfusion was thought of at too late a period to be of any aid to the patient.

Septic conditions, such as acute septic endocarditis, are not benefited by any transfusion. I have had many such cases transfused but with indifferent results.

I have related enough to show that there are a variety of conditions in infancy and childhood in which if transfusion is performed in a rational way it may be a life-saving operation. I have no patience with the indiscriminate use of transfusion. Many practitioners recommend transfusion as a *dernier ressort*. This should not be so. There should be a very definite idea or indication to the performance of transfusion if we do not wish to bring this whole method of therapy into disrepute.

In closing this paper I wish to call attention to some points which may escape analysis. Transfusion has its greatest success in those cases in which there is a real loss of blood from the blood volume, such as in the newly born, in melena or hemophilia; where the blood is not actually lost and the blood volume is not reduced we must be cautious in transfusing. Such are the cases of sepsis with hemolysis, as in the case of sinus thrombosis as related previously in this paper. In these cases we wish really to replace the existing blood with new blood and in such and similar cases transfusion should be preceded by preliminary blood letting. In no other way can we imagine large quantities of blood to be introduced into the system without effecting a most damaging strain. Many methods have been devised by which the amount of blood to be introduced could be calculated, but this is so wrapped up in mathematical formulas as to be most impractical. So that we are reduced to the safer method of removing a certain amount of infected blood from the system and then replacing it with fresh blood. We employ the same method in lumbar puncture, where an amount of subarachoid fluid is removed to be replaced by immunizing serum.

THE CALORIC REQUIREMENTS OF NORMAL INFANTS AND CHILDREN FROM BIRTH TO PUBERTY

FRITZ B. TALBOT, M.D.
BOSTON

During the past eight years Dr. Francis G. Benedict and I have been conducting a series of studies at the Nutrition Laboratory of the Carnegie Institution of Washington on the energy metabolism of normal infants and children from birth to puberty. A preliminary report of these investigations was recently given by Dr. Benedict in the Shattuck Lecture delivered before the Massachusetts Medical Society at Boston, June 3, 1919.[1] The detailed report of these observations will be published later in a publication of the Carnegie Institution of Washington and will contain a review of the literature of the metabolism of normal infants and children, and a complete discussion of the normality of the subjects studied. In this series of investigations we included 108 boys and 70 girls. The majority of the younger subjects were normal breast-fed infants. These were studied at the Boston Directory for Wet-Nurses. The older subjects were selected from the New England Home for Little Wanderers, all possible efforts being directed towards individuals who represented normality.

Photographs are shown and a description of the apparatus and technic used in these investigations is given in detail in the article by Benedict.[1]

Chart 1 gives the total basal metabolism for twenty-four hours of normal children from birth to puberty. In this instance the calories are plotted against age, showing that the total calories become larger as the age increases. By "basal metabolism" is meant the minimum metabolism necessary to maintain life with the patient at complete muscular repose, and, if possible, without any food in the stomach. It was impossible, however, to study the younger subjects without food. The older subjects were studied in nearly all instances without food.

Chart 1 shows that there is no difference between the basal metabolism of boys and girls up to about one year of age, or about 10 kg. of weight, after which there is a sex differentiation, boys of the same

1. Benedict, F. G.: Boston M. & S. J. **181**:107 (July 31) 1919.

weight having a somewhat higher metabolism than girls of the same weight until they reach 34 kg. of body weight, when the metabolism of both sexes is the same, after which the basal metabolism of girls is somewhat higher than that of boys.

If the basal metabolism shown in Chart 1 were doubled it would approximate the total calories that a child of a given weight would require. This, however, is not quite accurate because the growth factor is greater during the first year of life than it is during later childhood. It is necessary, therefore, to a little more than double the basal metabolism before the age of six months in order to obtain the total metabolism, whereas, after six months of age, doubling the basal

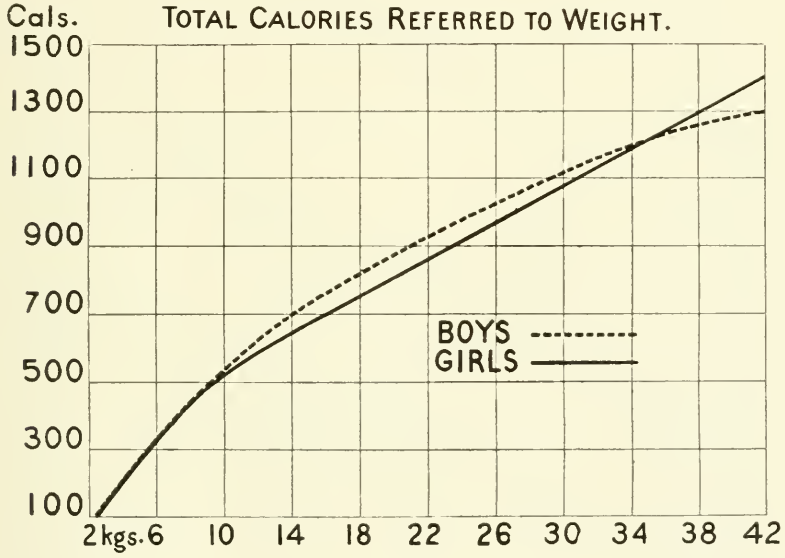


Chart 1

metabolism more than gives the total number of calories that are necessary for a child to grow. Whether these figures will be sufficient will depend upon the habits of the child, as shown by Captain Gephart at St. Paul's School in Concord, N. H., in which he found that the average intake in food in his subjects was 5,000 calories per day.[2]

Chart 2 gives the measured total basal metabolism of infants from birth to one year, and at the same time estimated curves showing the weight, food intake and available food.

2. Gephart, F. C.: Boston M. & S. J. **176**:17 (Jan. 4) 1917.

The lower line on Chart 2 represents the total basal metabolism, which, at six months, is 400 calories. This does not mean, however, that all normal children have a basal metabolism of 400 calories at six months of age, since some infants grow much more rapidly and attain

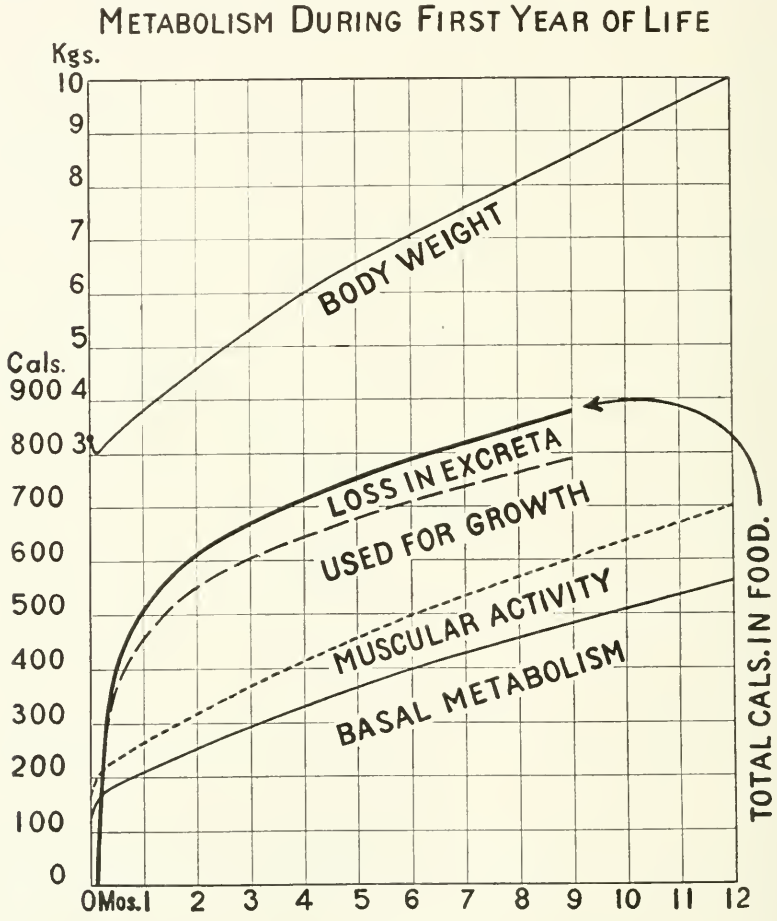


Chart 2

a greater length and weight than other infants of the same age. Infants with a greater weight than the average tend to have a higher basal metabolism than the curve given here, while normal infants with less weight than the average usually have a slightly lower basal metabolism.

The upper curve represents the normal increase in body weight of an average infant from birth to one year, and shows the initial loss of weight which occurs during the first three days of life, before the breast milk "comes in."

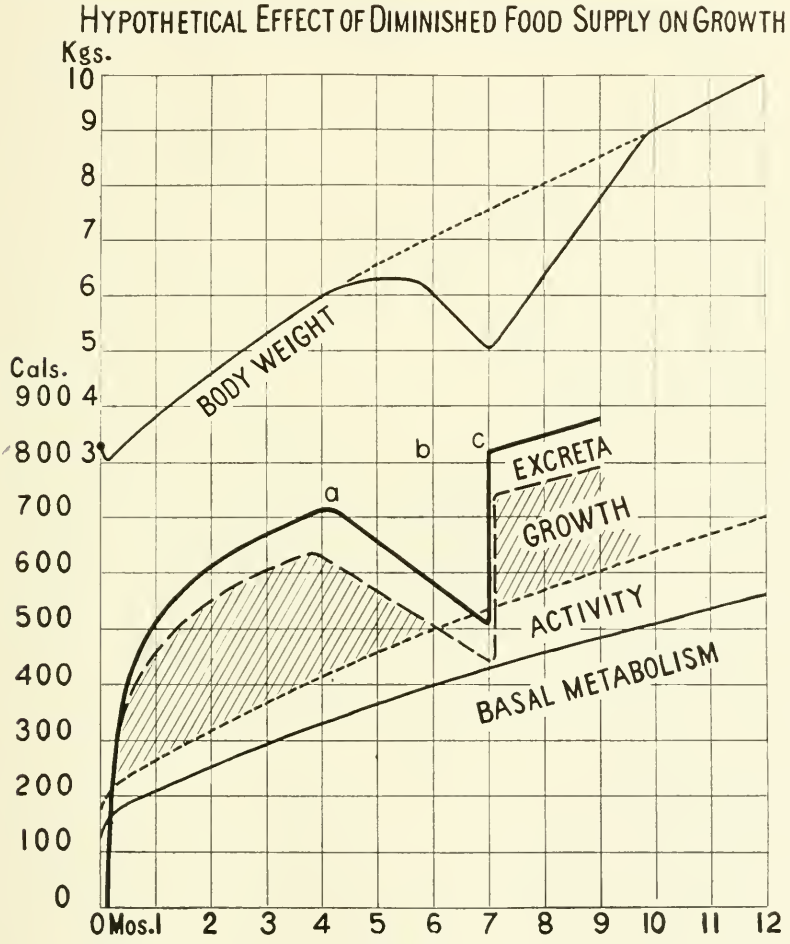


Chart 3

The heavy line to which the arrow points indicates the intake of breast milk, which commences about the third day and represents on an average the total calories normally received about this time. This rises rapidly after a sufficient amount of breast milk calories are sup-

plied for the infant to gain in weight. This curve, representing the number of calories in the breast milk, is estimated from figures found in literature. The total number of calories supplied in the food in this manner naturally vary with the different nursing mothers, and are

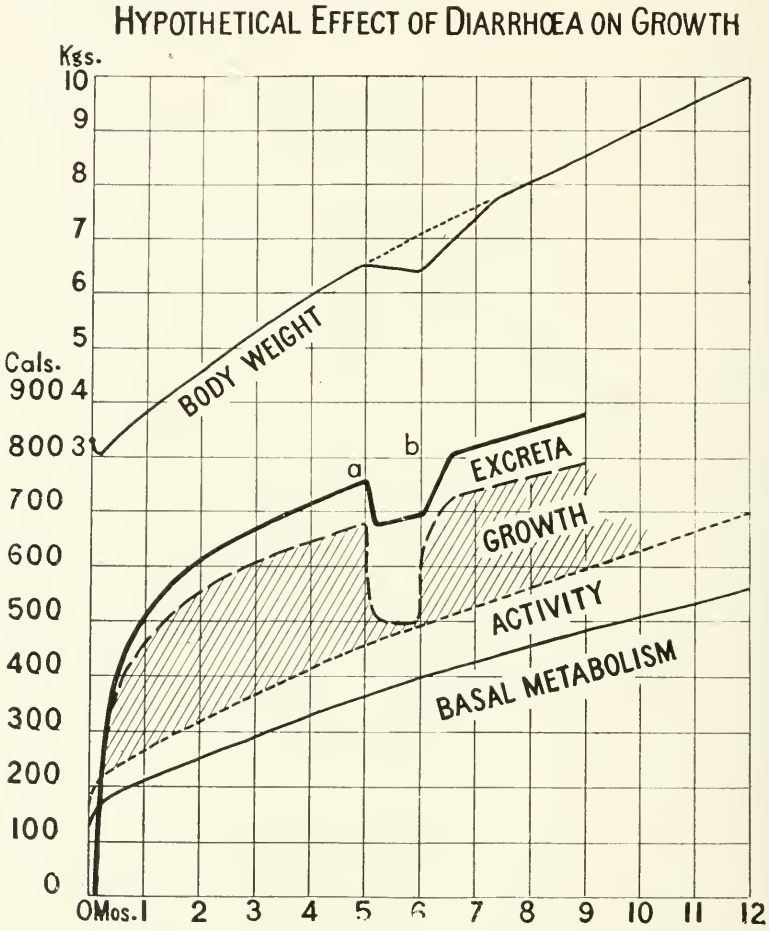


Chart 4

influenced both by the quality and quantity of the milk. It may be assumed generally that at the end of the first month a baby will be receiving, on an average, approximately 500 calories a day, and at the end of the ninth month about 880 calories a day.

It has been estimated that about 90 per cent. of the food taken by an infant is available for use in the body, the balance being lost in the excreta. Taking this as a standard, the curve in Chart 2 represented by long dashes gives the available energy which an infant may use at

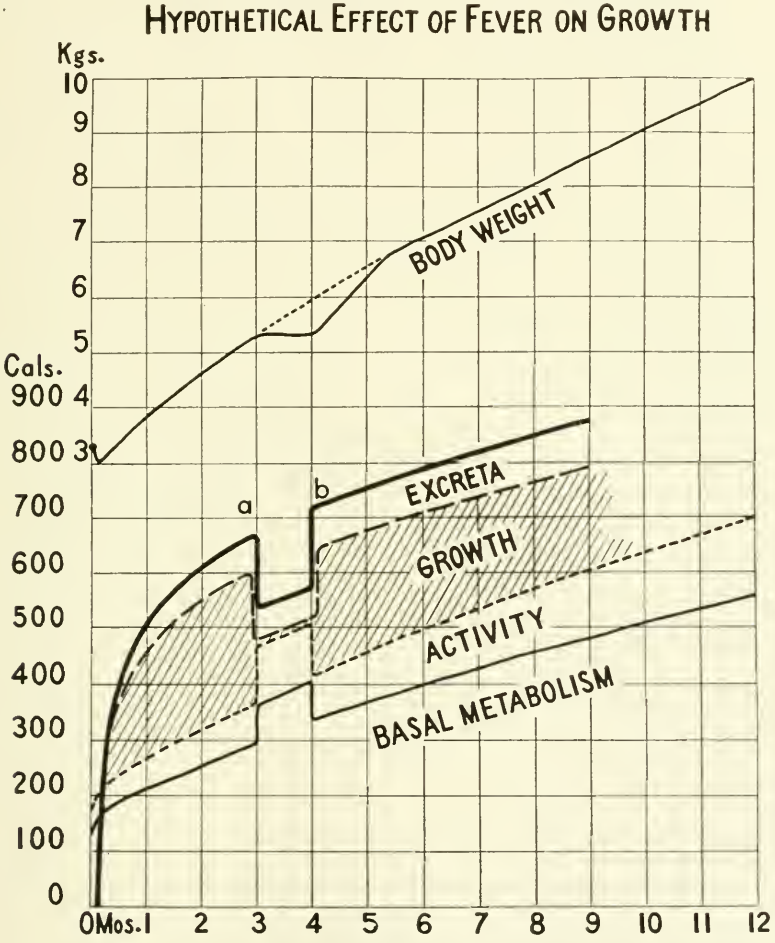


Chart 5

a given age. For example, at six months about 785 calories are ingested, about 705 of these calories being available for use in the body, the remaining eighty calories having been lost in the excreta.

It has been shown by other investigators as well as ourselves, that muscular activity increases the metabolism. We have shown that in new-born infants crying and kicking may increase the metabolism as much as 200 per cent. for a short time. To estimate the energy used

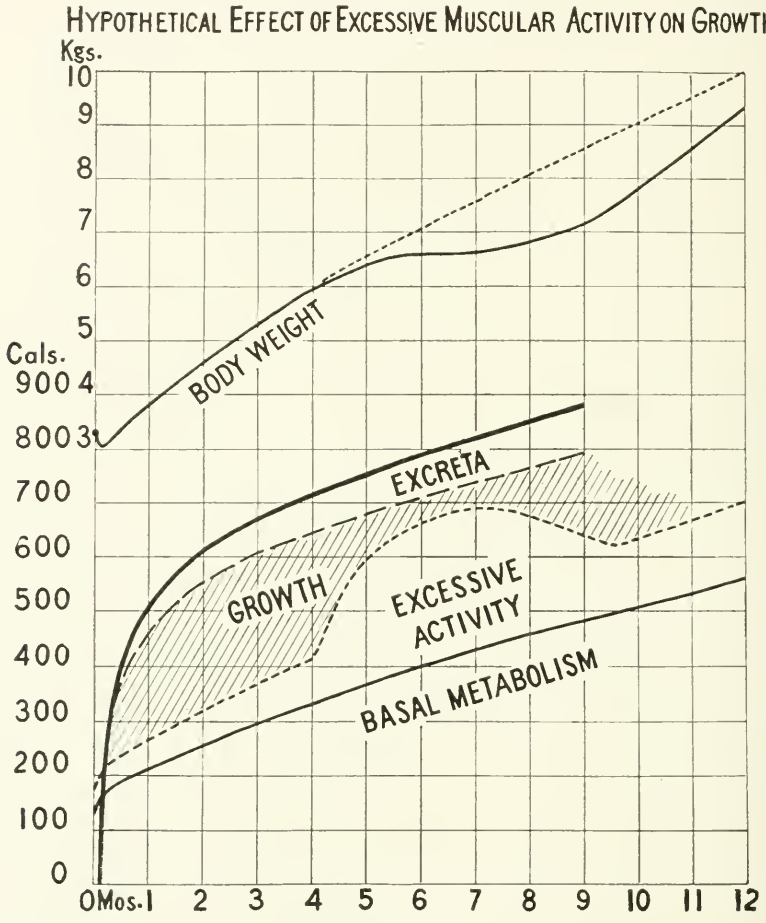


Chart 6

up by muscular activity throughout an entire day, continuous direct measurements are necessary. This was done in two normal infants previously reported by Talbot.[3] In one of these infants the increase

3. Talbot, F. B.: Am. J. Dis. Child. **14**:25 (July) 1917.

in metabolism throughout the twenty-four hours was 30 per cent., and in the other 20 per cent., over the basal metabolism. It may be assumed, therefore, until further figures are obtained, that the average increase of metabolism, due to muscular exercise, over the basal metabolism in a normal infant is 25 per cent. This percentage was, therefore, added to the basal metabolism and is represented in Chart 2 by the short dashes, and shows that at six months the basal metabolism of an infant is 400 calories with 100 more calories used up during the day in muscular activity. This figure, however, can only be taken as the average amount, since it cannot be assumed that all infants have the same amount of muscular activity.

The greatest rate of growth is during the first five to six months of life when an infant doubles its birth weight. During the second six months of life, the gain in weight is approximately the same as during the first five or six months, but, whereas, the increase in weight was 100 per cent. in the first six months of life, it is in the neighborhood of 50 per cent. in the second six months. Since the period of greatest growth is during the first year of life, relatively more food must be available for growth during this period than at any other time. It is obvious that the rate of growth is affected by the number of available calories. In Chart 2 the space between the lines of short and long dashes represents approximately the amount of food which goes for growth. According to the figures on this chart, which, it must be remembered, are only estimations, 36 per cent. of the food intake went for growth at three months; 26 per cent. at six months, and 21 per cent. at nine months. According to Cammerer,[4] only 15 per cent. of the food energy was retained for growth, while Rubner and Heubner[5] found it to be 12.2 per cent.

In Chart 3 is shown the hypothetical effect of a diminished food supply on growth.

According to Chart 3, an infant received sufficient breast milk for growth up to four months of age, to the point marked *a*, at which time the breast milk began to give out. During the course of the next three months the milk continued to diminish very rapidly, but the mother was unwilling to give a supplementary bottle feeding. At the point

4. Cammerer: See Lusk, The Science of Nutrition, Ed. 3, Philadelphia and London, W. B. Saunders & Co., 1917, p. 412.

5. Rubner and Heubner: See Lusk, The Science of Nutrition, Ed. 3, Philadelphia and London, W. B. Saunders & Co., 1917, p. 412.

marked *b* it is seen that the available energy just equals the calories extended by the basal metabolism and the muscular activity of the infant, and that nothing is left for growth. At this point the infant's weight is stationary. Between the points marked *b* and *c* the available energy is less than the amount used up by the infant in its basal metabolism and muscular activity. At this period the infant must obtain its food from some other source, and does so by using up its body tissue, thus losing in weight from 6 to 5 kg. Presumably, this is done at the expense of its own body fat.[6] At the point marked *c* the condition of the infant becomes so serious that the parents are willing to consult a physician and have a supplementary bottle feeding given. Enough calories are then added to bring the total food up to the normal requirements for growth, the infant gains rapidly, and after enough food is given and properly digested, it reaches the average weight for its age in a short time. Osborne and Mendel[7] have shown by their observations on white rats that it is possible to resume growth after long continued failure to grow.

Chart 4 shows the hypothetical effect of diarrhea on growth.

The recent work of Holt and his co-workers[8] shows that a great deal of fat is lost in the stools during diarrhea. Normally, 91.3 per cent. of the fat in the food is retained in the body, while in diarrhea only 79.3 per cent., and in severe diarrheas only 58.5 per cent. is retained. There is a corresponding loss of protein when casein curds are found in the stools. In one instance noted by Talbot and Gamble,[9] 24 per cent. of the ingested nitrogen was lost in the stools.

In Chart 4 it may be assumed that at the point marked *a* an infant commences to have diarrhea, and, as a consequence, there is a great increase in the number of calories lost in the stools, as shown by the chart, with the usual loss in appetite. The available energy is then reduced to such an extent that the infant has nothing left for growth and stops gaining. If the infant becomes restless, and the muscular activity increases, more than 25 per cent. more calories will be burnt up than are available for use, and the infant will lose weight. At the

6. Benedict, F. G., and Talbot, F. B.: The Physiology of the New-Born Infant, Carnegie Institution of Washington, Publicatoin No. 233.

7. Osborne, O. T., and Mendel, L. B.: J. Biol. Chem. **22**:439 (Sept.) 1915.

8. Holt, L. E., Courtney, A. M., and Fales, H. M.: Am. J. Dis. Child. **17**: 423 (May) 1919.

9. Talbot, F. B., and Gamble, J. L.: Am. J. Dis. Child. **12**:333 (Oct.) 1916.

point marked *b* the diarrhea stops, there is no longer excessive loss of energy in the stools, and growth is resumed.

Chart 5 shows the hypothetical effect of fever on growth.

It has been shown that a rise in body temperature increases the metabolism.[10] In Chart 5 it may be assumed that an infant becomes feverish at the point marked *a*, and there is an increase in the basal metabolism. Associated with fever is nearly always an increase in muscular activity and restlessness due to discomfort, with a loss of appetite and consequent drop in the caloric intake. This is shown on the graphic chart and the result is that during fever there is no food left for growth. If the available energy becomes less than the energy used up by the basal metabolism plus the muscular activity the child loses weight.

Chart 6 shows the hypothetical effect of muscular activity on growth.

Northrup[11] reported several cases of infants with nervous prostration in which there was excessive muscular activity due to prolonged mental stimulation. Such infants are continuously on the *"qui vive,"* are very active with the slightest provocation, sleep less than the normal, and practically always lose their appetite. The effect of excessive muscular activity on growth in such cases is illustrated in Chart 6. It also pictures what happens to the older child who overexercises and runs himself "as thin as a rail."

Chart 1 and the lower line on Chart 2, representing the basal metabolism, are the results of actual measurements of the metabolism of normal infants and children. The remaining curves on Chart 2 and Charts 3, 4, 5 and 6 were plotted merely to give a graphic picture of characteristic cases. They do not represent cases actually measured in real life.

DISCUSSION

DR. HOLT: This paper ought not to be passed by without at least some expression of appreciation on the part of this society. It explains a great many of our common experiences: for instance, individual differences in children so far as their food requirements are concerned. We know that it is impossible to feed a baby absolutely according to its weight or according to its age, and that a great many factors must be taken into consideration. It is only by taking all the factors into account that we can come to judge exactly

10. Lusk: Loc. cit., p. 499.
11. Northrup: Arch. Pediat. **22**:14, 1905.

how much food a child should be given. Dr. Talbot has put the different factors down in figures showing just how much is to be attributed to the different ones; this we have done heretofore but only very crudely. This is the first time these things have been presented to us in a scientific way so we can all see for ourselves exactly what happens.

The question of muscular activity in relation to weight-increase is very important, not only in infants but in older children. The problem we frequently meet in children is that of not properly gaining in weight. A child may gain only 1 or 2 pounds a year and the most important thing to do may simply be to reduce his excessive muscular activity. Some children seem endowed with a capacity for almost perpetual motion, and it is often impossible to secure any proper gain in weight unless we can reduce their activity. No matter how we may change the diet these children will not progress until we solve this part of the problem.

Another point which Dr. Talbot has called attention to is the large amount of food lost in the stools. We see this constantly in children who continue to lose weight though they are getting two or even three times as much food as they can take care of and the effort is being made to give even more food to make them gain in weight. How great the loss may be we do not realize without a chemical examination of the stools.

DR. TALBOT: I am very much pleased to have interested you, for it was with difficulty that we have been able to sift out these data.

Dr. Griffith has asked me about the energy quotient in different periods. If you take one of these babies weighing 6 kg. at 4 months of age his energy quotient would be about 111.

We can almost assume for purposes of simplicity that we can make one line and not separate boys and girls as the differences are very slight. If we double the basal metabolism we would get approximately the number of calories the child would need. I think probably the greatest importance of this chart will be in the instruction. If a series of charts is made with this as a basis the visible figures will fix in the student's mind the caloric side of infant feeding. It seems to me that these charts can well be taken to show where the food goes and why it goes to certain places.

POSTINFLUENZAL ENCEPHALITIS *

HENRY HEIMAN, M.D.
NEW YORK

INTRODUCTION

The pandemic of influenza which in the fall of last year spread with such alarming virulence over two continents spared no age. Though perhaps less frightful in its ravages among infants and children, there were many serious complications. Among these was a group of very unusual symptoms related to the central nervous system. Sudden onset, two or three weeks after an attack of influenza, of stupor or coma, paralysis with frequent cranial nerve involvement and a normal cerebrospinal fluid were the most significant manifestations. A study of eight such cases from the Pediatric Service of Mount Sinai Hospital form the basis of this paper.

HISTORY

To modern observers, it seemed for a while to be a new symptom complex, but as early as 1712, during an epidemic of influenza, Zuelzer[1] made note of peculiar drowsy states complicating many cases. In the course of the extensive epidemic of 1890 numerous references were made to similar nervous conditions. Henoch[2] described a case of polioencephalitis with paralysis of one arm which developed in a girl, 11 years of age, two weeks after an attack of influenza. Remak[3] noted a fatal case of bulbar paralysis in a child immediately after influenza. A case of stupor, convergent strabismus and hemiplegia following influenza in a child, 3½ years of age, was described by Knotten.[4] The same author notes a case of polioencephalitis in a

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1. Zuelzer: Influenza, Ziemssen's Handb. d. Speziellen Pathol. und Therap. **2**:506, Part 2, 1874.

2. Henoch: Berl. klin. Wchnschr., No. 8, 1890.

3. Remak: Quoted by Bosseks; Die Geschichte der Influenza und ihre Nervöse u. Psychische Wachkrankheiten. Leiden, E. Ijdo, 1894.

4. Knotten: Quoted by Kohts; Ueber Paralyse u. Pseudo-Paralyse im Kindesalter nach Influenza.

child, 4½ years of age, during the epidemic. Alfred Brionne[5] refers to a case of polioencephalitis with paralysis of deglutition, reported by Uhthof. Otto Leichtenstern,[6] in 1896, was the first to give a complete detailed account of the condition. Following the recent epidemic a large number of cases were observed in England and France. An exhaustive report was presented before the Royal Society of Medicine last year. F. G. Crookshank[7] gave statistics of 127 cases that came under his observation in the London and North West London Hospitals. Fifty-seven of these cases occurred in persons under 10 years of age. Drowsiness was noted in thirty, stupor or prolonged lethargy in twenty-six, "unconsciousness" or coma in twenty-five cases. Paresis or paralyses of limbs were present in forty-seven cases and ptosis was marked in twenty-three cases. Neal[8] described cases occurring among children in New York.

TERMINOLOGY

The first term applied to this syndrome was "sleeping sickness" as described in the epidemic of 1712 in Tübingen by Zuelzer. Such a nomenclature would confound the condition with trypanosomiasis. During the epidemic of 1890 reference was frequently made to nona, a meaningless term, which was soon forgotten. "Grippal encephalitis" was used by Leichtenstern. "Lethargic encephalitis" was suggested by Economo[9] in 1917, but this has been criticized by Bassoe[10] as being illogical as the patient not the encephalitis is lethargic. He proposes the term "epidemic encephalitis." We believe from a study of the relation of this condition to influenza that the most appropriate designation is postinfluenzal encephalitis.

ETIOLOGY

One cannot fail to be struck by the close association of this symptom complex with epidemics of influenza. The only periods during which it has been observed have been the years following the extensive

5. Brionne, Alfred: Contribution a l'étude de la forme nerveuse de la grippe et de ses complications. Paris, H. Jouve, 1890, 62, p. 14, No. 378.

6. Leichtenstern, Otto: Influenza and Dengue, Wien., Nothnagel **4**:118, 1896.

7. Crookshank, F. G.: Brit. M. J. **2**:489 (Nov. 2) 1918.

8. Neal, Josephine B.: Meningeal Conditions Noted During the Epidemic of Influenza, J. A. M. A. **72**:714 (March 8) 1919.

9. Economo: Wien. klin. Wchnschr., July 26, 1917.

10. Bassoe: J. A. M. A. **72**:972 (April 5) 1919.

epidemics. Pfuhl[11] has been able to isolate the influenza bacillus from the brain of one of these patients. This finding, however, has not been confirmed. All our own cases were preceded by attacks of influenza.

PATHOLOGY

Leichtenstern, in 1896, described the condition as a hemorrhagic encephalitis. A most careful and detailed account of the pathologic changes has been contributed by M. Marinesco[12] at Bucharest. He examined two specimens. Grossly disseminated punctiform hemorrhages in the gray matter about the floor of the fourth ventricle, the acqueduct of Sylvius and in the posterior part of the pons and the peduncles were found. The cerebral cortex showed only congestion of some of the vessels of the leptomeninges. Microscopically, he found four kinds of lesions: 1. Infiltration of the walls of the small vessels, especially the veins with lymphocytes and plasma cells in the adventitia in several layers. 2. Foci of interstitial inflammation formed of neuroglia cells and large cells with eccentric nuclei, lymphocytes and polynuclears. 3. In some of the nerve cells there was dissolution of Nissl's bodies, relative achromatosis, reduction in volume of cell body and of the number of prolongations. 4. Numerous foci of hemorrhages around the walls of the small blood vessels. We were unable to make any postmortem pathologic examinations as none of our patients succumbed to the disease.

SYMPTOMATOLOGY

From our own observations of the cases we were able to differentiate three main forms, depending on the severity and the most prominent symptoms. We may group them into the (1) irritable, (2) lethargic and (3) lethargic with paralyses. The irritable type is characterized by marked restlessness, excitability and almost continuous crying.

A girl, three years of age, was admitted to the hospital with a history of influenza two weeks previously. Four days before admission the mother noticed the onset of spells lasting about one-half hour and appearing every three or four hours during which the child would cry and shout in wild excitement. This continued for several days in the ward. There were no convulsions and no paralyses. The

11. Pfuhl: Berl. klin. Wchnschr., p. 979, 1892.
12. Quoted by Lieut.-Col. F. W. Mott, Brit. M. J. **2**:489 (Nov. 2) 1918.

temperature ranged between 97.8 and 101 F. The blood count showed a moderate leukocytosis, with 13,800 leukocytes; polymorphonuclears, 74 per cent.; lymphocytes, 25 per cent.; transitionals, 1 per cent. The cerebrospinal fluid was normal. The patient recovered completely in ten days. This represents the mildest form.

In the lethargic form the most prominent feature, as the name implies, is a drowsy state. The facial features are expressionless, masklike, resembling the Parkinson syndrome. The patient lies in a semicomatose condition for weeks, resents being aroused and takes nourishment with great difficulty. There may be convulsions or twitchings of the face muscles. There is frequently moderate rigidity of the neck. Most of our cases fell into the third group of lethargy with paralyses. In this type we find, in addition to the stuporous state, some form of paralysis and frequently convulsions. There may be an involvement of the extremities or cranial nerve palsies.

Age.—The youngest patient observed by us was four months of age, the oldest thirteen and a half years; three patient were under one year of age; four were under three years.

Sex.—In our series the cases were equally divided between the two sexes. Crookshank's cases included thirty-three males and twenty-four females.

Mode of Onset.—In all our cases there was a history of previous influenzal infection. In one child the nervous syndrome followed immediately, and in another four months had elapsed. The average interval was about two weeks. In six of the cases the onset of the encephalitis was sudden, in two there was a gradual progressive development. Convulsions were the first manifestations in three cases, stupor in four, marked irritability in one.

Physical.—In half the cases there was some degree of rigidity of the neck. Facial paralysis was present in three, difficulty in deglutition in two cases. In four cases there was weakness of the neck and back muscles, making it impossible to hold the head erect or to sit up. There was a spasticity of the lower extremities with increased knee jerks in three, twitching in one case. One case presented a bilateral internal strabismus, another marked nystagmus. Drowsiness or stupor continued as a prominent symptom throughout the course of the illness in four cases, irritability in three. The masklike expression was present in a more or less degree in the majority of our cases.

Temperature.—Fever has played but a small rôle in all of the cases reported. Rarely has there been a rise of more than two or three degrees above normal. In one of our cases it was as high as 102 F., in another 101 F. The remainder ranged below 100 F. The pulse and respirations were not unusually high.

Fundi.—The fundus of the eye was examined in all of our cases, but showed nothing characteristic. In a few instances there was slight pallor of the disks. In one case optic atrophy developed late in the disease.

Cerebrospinal Fluid.—Of the eight specimens examined, only one showed definite changes. There were eighty cells per c.mm., 80 per cent. of these being lymphocytes. Albumin was present, forming a 1 mm. ring. Fehling's reaction was present. No organisms were found. One other case showed a somewhat increased pressure. The remaining specimens were all within normal limits. In a case reported by Ely,[13] anaerobic as well as aerobic cultures of the fluid were negative. Among Crookshank's cases over 80 per cent. showed some increase in the cell count.

Blood Counts.—Marked leukocytosis was present, in one case being as high as 34,000 cells, with a differential of 84 per cent. polymorphonuclears, 12 per cent. small lymphocytes and 4 per cent. large mononuclears. In two cases a normal blood picture was present, and the remainder showed a moderate leukocytosis ranging between 9,000 and 13,800.

PROGNOSIS

When the cases first appeared with the marked lethargy and frequent paralyses, the prognosis suggested was usually bad. But we have found it much better than the alarming state of the patient seemed to indicate. Thus far, we have not lost any cases. In at least two cases, however, the mentality has been definitely affected. The children have become imbecilic. In this respect the condition differs from the polioencephalitic form of infantile paralysis in which the mentality is rarely ever markedly changed. In three others the paralyses, though considerably improved, have not yet disappeared completely. The irritable type of case recovers rapidly.

13. Ely: J. A. M. A. **72**:985 (April 5) 1919.

TREATMENT

There is no specific therapy. Our treatment has been symptomatic throughout. For the irritable type mixed bromids and chloral are of some value. In the paralytic type massage of the involved muscles is of distinct advantage. Good results have been reported from lumbar punctures.[13] We have not found this procedure of special benefit in our therapy.

64 West Eighty-Fifth Street.

DISCUSSION

DR. RUHRÄH: The history goes back farther than Dr. Heiman has given it. Hippocrates has mentioned the condition. From the sixteenth century on there are numerous very good descriptions. The condition was described under various names. In Germany it was referred to as the Schaffkrankheit, which, in 1712, was changed intentionally or accidentally by Camerarius to Schlafkrankheit, a much better name. It was also described under the name of Raphania by Röthman, a pupil of Linné, as he thought it was due to radish seeds in the threshed grain. In 1755 it was described as sausage poisoning. There is a very interesting article on this subject by F. G. Crookshank, in the Proceedings of the Royal Society of Medicine, 1919, Volume XII (Section of the History of Medicine), pp. 1-21.

DR. KOPLIK: I do not care to let this paper go by without pointing to the fact that throughout the epidemic of influenza in New York we all saw these cases and the clinical features have been well described by Dr. Heiman. They are similar to cases of poliomyelitis or polio-encephalitis and have been observed on both sides of the Atlantic. They seem to be of the hemorrhagic type and to have affected the brain and not the cord. The nerves were affected as in polio-encephalitis. There were cases with ophthalmoplegia, facial paralysis, a rigid neck, Macewen and Kernig signs, all quite marked. Many of the mothers whose children had this type of the disease felt very uneasy. The lethargic cases resembled tuberculous meningitis and some physicians made the diagnosis of tuberculous meningitis to find in a week or ten days that their patients were on the road to recovery.

In some of these cases we punctured, but we found that this type of cases did well without lumbar puncture. In one case that I recall the diagnosis of tuberculous meningitis was made. We waited a little and did not puncture and the child got well without puncture.

There were different types of encephalitis following influenza; they were not all lethargic cases, but all types of encephalitis were found.

DR. ABT: All the cases of encephalitis we have been seeing are not due to influenza. One frequently sees encephalitis with pneumonia, measles, typhoid fever and even with whooping cough. Encephalitis constitutes a group of diseases, as Dr. Koplik has pointed out. The classical description is that of Sigmund Freud. The disease is selective, selecting certain portions of the brain. There are a number of cases in which the anterior motor areas are affected, and in some of these there is unilateral paralysis. The prognosis is good so far as life is concerned, but a large number of those affected remain paralyzed.

DR. SMITH: I wish to report the case of a child with encephalitis which began at the age of 2 months and has now lasted four months. There was facial paralysis and inability to cry. The facial paralysis is much better, but the child is still unable to cry aloud. At the onset of the illness there was contraction of the pupils which, associated with lethargy, led to a suspicion of morphin poisoning. There was no history of influenza in this baby and at entrance the roentgenogram of the chest was negative. Finally, however, there was a bronchopneumonia in the right chest.

DR. TALBOT: I would like to ask Dr. Heiman whether he has any reports from adults as to whether double vision was among the early symptoms.

DR. HEIMAN: I want to thank Dr. Ruhräh for the information he has given me with reference to the early history.

We know that this is nothing new because there are certain strains of bacteria of the infectious diseases that have a selectivity for the nervous system. We have seen it in typhoid and other infectious diseases. Among the 25,000,000 cases of influenza that occurred, there were of course many of these cases seen. We made the diagnosis postinfluenzal, because all of our cases were preceded by influenza. Our youngest case was in a child 4 months old. We had no case with diplopia. We have seen ophthalmoplegia.

THE CAUSE OF A "SPORADIC" CASE OF CEREBRO-SPINAL MENINGITIS

HENRY L. K. SHAW, M.D.
Clinical Professor of Diseases of Children in the Albany Medical College
ALBANY, N. Y.

This case is presented as a contribution to the epidemiology of cerebrospinal meningitis. A 5-months-old breast fed infant living on a farm developed cerebrospinal meningitis. There was no record of any previous cases in the locality. On account of the isolated location of the farm it was possible to ascertain and examine every person who had come to the house for a week previous. One of the visitors was the uncle of the baby, a man, 33 years of age, who enlisted in the army in April, 1917, and went to France with the Fifty-Second Coast Artillery in August, 1917. He returned to this country in January, 1919, and was discharged from Camp Upton, Jan. 27, 1919. He fondled the baby January 28. A few days later the baby was taken ill and I was called in consultation February 3 by Dr. Reid of Coeymans. Examination of the spinal fluid confirmed the diagnosis of meningococcus meningitis.

The returned soldier said that to his knowledge he had never seen or been in contact with or known of a case of meningitis in his regiment, and had never been sick himself. Miss Stewart, of the New York State Department of Health Laboratory, took postnasal cultures from eight persons which were plated at the farm. All were negative except those from the soldier, and the meningococci obtained from his were agglutinated by the fluid obtained from the spinal fluid of the baby and were also proved to be identical by culture. A point of interest is that the organism still persists in this carrier (June 10). He has used irrigations of all kinds and has been under the treatment of a nose and throat specialist, but the meningococci resist all bacterial procedures. His plight is pitiable. The army refused to take him back. The War Risk Insurance Bureau, the U. S. Public Health Service and the Red Cross all claim they have neither funds nor authority to reimburse him. He is in perfect health, but is held in quarantine and not able to support himself or his family.

All of the textbooks on diseases of children which I have been able to consult mention that cerebrospinal meningitis appears sporadically or in epidemics, implying that there are two forms of the disease. Kohls[1] states that the disease has a sporadic tendency without being confined to the region of its development. Zappert[2] alludes to the possibility of animal carriers. "If distributed sparsely they, of course, appear to individual observers as sporadic cases and not actually to be connected with an epidemic. Perhaps the germs are carried by domestic animals and also by vermin."

Holt and Howland[3] sum up the opinion of pediatricians, prevailing in 1914, when the sixth edition of their textbook appeared, as follows: "Sporadic cases of meningitis occur after epidemics and quite apart from them without apparent cause, and it is very exceptional that any connection with a previous case can be established."

The question arises as to what is a sporadic case of disease. In the strict interpretation of the word, there is no such thing, but the term is used to denote isolated or untraced cases. Up until a very short time ago two theories were accepted to account for the appearance of these cases. Infection by or through the air from distant points and by fomites. Scientific investigations and public health experience have disproved the theory of aerial infection. By fomites we do not include instances which are by no means uncommon where there is a direct transference of germs from one person to another on such objects as pencils, cups, fingers, etc., for this is really contact infection. Infection by fomites means by infected objects which retain the infection for a long period of time and over great distances and later give rise to the disease. The experimental work on yellow fever in Cuba which was carried on by Reed and Gorgas proved conclusively that infection could not be transmitted by fomites. The results of investigations of other communicable diseases all point to the fallacy of the fomites theory. Cases which apparently have been traced to fomites can always be traced further to a previous case if full facts are obtainable. Lack of appreciation of modes of infection, both by the laity and medical profession, is responsible for failure to obtain full facts.

1. Deutsche Klinik.
2. Pfaundler and Schlossman's Diseases of Children.
3. Holt and Howland.

Charles V. Chapin[4] says that "laboratory investigation shows that fomites infection with spore bearing bacteria is common; that such infection in typhoid fever, tuberculosis, diphtheria and with other resistant organisms doubtless sometimes takes place; that it is possible in chorea and plague, while such infection in gonorrhea, influenza, cerebrospinal meningitis and pneumonia must be practically impossible."

It is difficult to make the public and even members of the medical profession realize that persons, not things, are responsible for the transmission of communicable disease. Many instances of alleged fomites infection were doubtless cases of carrier infection. Persons in whom the germs are growing are much more likely to be the agents of infection than are objects on which the germs are dying.

Hill[5] sums up the present views as follows: "The old public health was concerned with the environment, the new is concerned with the individual. The old sought the sources of infectious diseases in the surroundings of man; the new finds them in man himself. The old public health sought these sources in the air, in the water, in the climate and topography of localities, in the temperature of soils at the depth of four and six feet, in the rise and fall of ground water, and failed because it sought them, very painstakingly and exhaustively it is true, in every place and every thing where they were not. The new public health seeks these sources — and finds them — among those infected persons (or animals) whose excreta or other constituents or body contents enter the bodies of other persons." The old public health also failed to find the routes of transmission of communicable diseases.

The widespread epidemic of poliomyelitis in the summer of 1916 gave abundant opportunity for the study of its epidemiology. Flexner and others were able to definitely prove that healthy carriers could and frequently did transmit the disease. The existence of such carriers had been suspected as otherwise the epidemiology was inexplicable. The striking similarity between poliomyelitis and cerebrospinal meningitis in the mode of transmisson, portal of entry and contagiousness has been pointed out by many investigators.

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4. The Sources and Modes of Infection.
5. The New Public Health.

In this connection it is of interest to note that Kerley[6] in 1916 explained the occurrence of poliomyelitis in isolated and remote distances by healthy carriers and came to the conclusion that the disease may be communicated by personal contact and spread by carriers.

Heiman,[7] in 1917, emphasized the fact that poliomyelitis was spread by innocent carriers. Chapin[8] states that observations by different authorities indicate that a very considerable number of persons who come in contact with cases of cerebrospinal meningitis may carry the organisms on their mucous membranes, yet very few of these persons become infected. He states further that cerebrospinal meningitis extends almost entirely through the agency of carriers.

A carrier is a person who harbors disease germs in his body and is capable of infecting others directly or indirectly. There are two types of carriers, active and passive. The active carrier is one who has had the disease but continues to harbor the organisms after clinical recovery. The passive carrier is one who has been in contact with a patient but has at no time suffered from the disease. A more accurate and descriptive nomenclature would be to designate the two types as convalescent and contact carriers. It is not essential, as far as the virulency of the organism is concerned, to distinguish between the two. Simon[9] estimates an average of ten carriers to a patient and as carriers beget carriers it is manifestly impossible to discover them all. The passive or contact carriers are a greater source of danger as their movements are unrestricted and they can disseminate disease over an unlimited territory.

With the advance of scientific research our old pet theories go on the scrap heap while new thories are evolved. Now that transmission of disease through the air and by fomites has been disproved, the present explanation is that communicable disease is transmitted only by means of infected persons, unrecognized cases and healthy carriers.

Epidemiologists claim that an outbreak of communicable disease can be checked with prompt and strict isolation of the sick and early and diligent hunt for carriers, but that after the disease has become established and carriers become numerous isolation and quarantine are of little avail. We were fortunate in being able to detect the carrier

6. Kerley: Arch. Pediat. **33**:424 (Jan.) 1916.
7. Heiman: Trans. Am. Pediat. Soc., 1917.
8. Chapin: Loc. cit.
9. Human Infection Carriers.

and to isolate both the patient and the carrier, and no further cases have developed. If, however, this carrier lived in a large city and had a large number of nieces, nephews and admirers, it is not difficult to imagine a wider distribution of cases with a large number of unrecognized carriers. Isolation, quarantine and disinfection of the sick would not have reached or controlled the situation. We lock the barn door after the horse and everything else has been stolen.

As the disposition and treatment of carriers is very largely a public health measure, a questionnaire was sent to the various state departments of health in order to ascertain their procedure regarding carriers of meningitis.

PROCEDURE REGARDING "CARRIERS" OF MENINGITIS IN THE VARIOUS STATES

Alabama .....Isolation until swabs negative. Strict isolation until well.

Arkansas .....Isolate until two successive negative examinations are secured. We have not had persistent carriers in this state, except in one or two instances where carriers were released by federal authorities from other states. In that instance no supervision whatever is exercised. One case in question was placed under observation with negative developments.

California .....No account is taken of meningitis carriers.

Colorado .....Owing to the small number of cases of this disease the problem of carriers has not come before us.

Connecticut .....Shall be isolated until examination of the nasal and throat discharges show the absence of the specific diplococcus.

Florida .....None at present.

Georgia .....No answer to this question.

Idaho .....Nothing relative to carriers.

Indiana .....Isolate them, etc.

Kansas .....Any person afflicted with this disease shall be isolated and quarantined for a period of not less than two weeks nor more than three weeks from onset unless temperature has not returned to normal in the meantime.

Kentucky .....Isolated during an epidemic.

New Orleans.....Isolation until three negative reports, when possible.

Maryland .....Outside of Baltimore City carriers of meningococcus are not looked for as a routine nor are quarantine regulations issued by the state.

Massachusetts ...Under the law the local board of health is directed to make such rules and regulations as are deemed necessary for the control of communicable disease, and our function in the matter would be advisory. We are pleased, of course, where it is practicable, to examine for carriers, and when found to advise as to procedure to render the nasopharynx free from meningococcus.

Minnesota .....None.

Missouri .....No statutory authority. At present time our statutes do not give the state board of health any authority concerning communicable diseases.
New Hampshire..Quarantine.
New Jersey.....Isolation of known cases required. No definite regulations concerning "carriers."
New York.....Contacts excluded from schools and gatherings; and if patient is not isolated, others in the household are quarantined. Carriers subject to special rules and regulations of the state department of health. Patients isolated until three successive cultures from the nasopharynx at intervals of not less than five days, are free of meningococci.
Oklahoma .....Isolation of infected persons and carriers until the nasopharynx is free from the infecting organisms, or at the earliest until one week after fever has subsided.
Pennsylvania ....Wherever we have knowledge of a meningitis carrier we keep the patient isolated until bacteriologic results are negative.
Rhode Island.....No action taken.
South Carolina...Quarantine carriers and actual cases for three weeks.
South Dakota....Every doubtful case must be classed as of epidemic type and cared for accordingly until proven otherwise.
Texas .....No regular line of procedure has been established.
Vermont .....So few cases in the state recently that it is difficult to make any report on it.
Virginia .....Quarantined when found.
West Virginia....Have had no occasion to do anything. Procedure same as in poliomyelitis.

The answers reveal very little uniformity in the isolation of patients and in dealing with carriers. There are no provisions for the detection of healthy carriers and the compulsory bacteriologic examination of contacts. Public health legislation on this subject is imperative if we are going to limit the spread of communicable disease.

New methods in the control and prevention of communicable disease are bound to result from the change in our knowledge of their transmission. The new public health recognizes the importance of contact and the significance of carriers. The old order of quarantine and disinfection is passing away and a new order of strict personal cleanliness and destruction of body secretions and excreta is at hand.

It is our duty as pediatricians to keep abreast of public health progress and to assist in educating our communities in the new order of things.

From the study of this case we feel justified in claiming that it was caused by contact from a healthy passive carrier, that sporadic cases are in reality untraced cases and that the human carrier is responsible for cases appearing either singly or in epidemics.

DISCUSSION

DR. HOLT: I would like to parallel Dr. Shaw's case. A young man returned from the army to his wife and 2-weeks-old baby. In a few days the wife developed a severe attack of typical cerebrospinal meningitis. The father was examined at a well known laboratory and found to be a carrier of the meningococcus, though he had not had the disease. After prolonged efforts to get rid of the organisms by sprays and douches he was recommended to take a long sea voyage. This he did, being gone nearly two months, but when he returned he was found to be still a carrier. Afterward he was placed under the care of an excellent nose and throat specialist, had adenoids and I think tonsils also removed and finally got rid of the organisms, whether permanently, it is as yet too soon to say.

It is important that we should recognize that many thousands of discharged soldiers are being distributed through the country and that undoubtedly many of these men are carriers and consequently we are certain to see many cases of meningitis develop whose origin it is difficult to trace. The problem is what to do with these individuals who are carriers, and what to do for them is an important one and has not yet been solved.

DR. KOPLIK: To show how vast and almost hopeless this problem is, Dr. Park undertook to find what the prevalence of diphtheria carriers in the public schools was. In one school he found that a good percentage of the children were carriers of diphtheria bacilli. This new test, the Schick test, is positive in a certain percentage, so that it is possible to pick out those who are susceptible to diphtheria. But it was found impossible to care for all the carriers. If we are going to isolate the carriers of all infectious diseases we will face a most difficult problem.

I had a case almost identical to Dr. Shaw's. A lady whose husband was abroad was blessed with a baby, a handsome, beautiful child. The baby was breast fed and nobody saw that child, except one person who came home from camp, visited the lady and brought her letters from her husband. A short time afterward the child had cerebrospinal meningitis, preceded by pneumonia and confirmed by lumbar puncture. This is similar to Dr. Shaw's case as showing the possibility of the case being due to a carrier.

DR. HEIMAN: I have seen the report of a baby, 6 weeks old, that was in the hands of a nurse, and never left home, yet developed meningococcus meningitis and died. Meningococcus meningitis is endemic in New York. It has been our custom to take two nose and throat cultures at an interval of three days, so that we can label the carriers. We cannot, however, do very much for them, as it is very difficult to get rid of the organisms.

We are now beginning to see the trench mouth due to Vincent's bacillus, an infection that is being brought home by the soldiers.

THE TERTIARY CEREBRAL MANIFESTATIONS OF HEREDITARY SYPHILIS

ALFRED HAND, Jr., M.D.

Following is the history of a patient referred to me by Dr. John H. Girvin in July, 1916:

R. H., boy, aged 8 years, is an only child; the parents have separated and the father is obtaining a divorce on the grounds of infidelity. The boy was breast fed during infancy; the aunt who is caring for him at present can only add the items that his tonsils and adenoids were removed two years ago, that he had a mild attack of measles eighteen months ago, and that the spells for which he is brought began three months later. These spells are described as attacks of cramps, occurring usually at 5 a. m., beginning in the left foot and spreading all over the body, with twitching; the jaws are set, the tongue is usually not bitten; relaxation occurs in a short time, and the boy feels tired after it. It is not known whether the attack occurs during sleep or after waking, but when the attack comes on, as it occasionally does, during waking hours, it can sometimes be checked or lessened in severity if firm pressure is made promptly around the left calf, before the cramp has spread above this; the boy has a warning of the attack, and tries to check it himself but does not succeed.

Examination showed a well-developed boy with an intelligent expression; height, 4 feet 2¾ inches; weight, 58 pounds; pupils equal and react to light and distance; tongue protrudes straightly; lungs and heart clear; palpation of abdomen negative; knee jerks exaggerated; Babinski and Kernig negative; no ankle clonus. Hemoglobin, 95 per cent. (Talqvist); red cells, 3,960,000; leukocytes, 7,000. Urine examination negative for albumin, sugar and casts.

The eyes were examined by Dr. H. Maxwell Langdon, who found hyperopic astigmatism, needing glasses.

Nothing abnormal was found in the ears, nose and throat by Dr. George B. Wood. Dr. Spiller examined the patient and was of the opinion that the attacks were probably true epilepsy. There was no regularity in their occurrence, nor could they be attributed to any special article of diet. After coming under observation on July 12, 1916, the dates of attack were July 14, 19, severe; 27, August 1, fell out of bed in this one and had to be lifted back; between August 18 and 26 six spells occurred, so the dose of strontium bromid was increased and the intervals between the spells lengthened to two weeks.

In October he started school, which made him a little nervous at first, but he soon quieted down; his weight showed an increase of 4 pounds and his height 1¼ inches in the three months; an attack occurred October 16; again on the 30th, the latter two days after a children's party. November 8, after a week of nervousness and irritability, there was a severe attack at 5 a. m., with much weakness, so that he had to stay in bed for a day; and for several days afterward there was a burning sensation like a belt around the abdomen with pains in both thighs. It was now noticed that he dragged his left leg. November 21, early, a severe cramp was associated, so it was reported, with unconsciousness, lividity and frothing at the mouth. Examination at this time

showed exaggeration of the knee jerks with ankle clonus; there was pain along the spine on bending over, but no prominence of a vertebra could be found; a dragging of the left leg in walking was apparent. A Wassermann test of the blood was made by Dr. Pfeiffer with a negative result. Administration of the bromid, which had been interrupted, was resumed, another spell occurring on November 23, but not again until December 6, 13 and 31 and January 1, a general nerve tonic (glycerophosphates and cod liver oil) being given.

In January, Dr. Pancoast made roentgenograms of the head, no abnormality being detected in the brain, but a study of the plates of the teeth by an orthodontist showed much impaction of the molars in the upper jaw, so the process of straightening was begun. Dr. Wood had called attention to the eruption of only three of the lower incisors on the lower jaw, but the roentgenogram revealed the fourth in place with plenty of room to come up. In February while in the house with chickenpox, no medicine was given and no spells occurred for nine days. April 6, the report was that since the orthodontia was started he had one bad spell with unconsciousness in the first week (bromids being given); three in twenty-four hours in the second week; two in one night in the third week; eight in the fourth, eight in the fifth and twelve in four days of the sixth week. A mixture of the triple bromids with adonis vernalis was tried without effect, the spells beginning to appear in the daytime and those at night being preceded by a cry.

He was then taken to the Children's Hospital for observation, the attacks continuing to increase until they reached a total of twenty-four in twenty-four hours. Another Wassermann test with a negative result was made, Dr. Kolmer doing our hospital work at this time. Dr. Burr studied him with me here and April 20 we had the opportunity of seeing one of the seizures; with the cry, "There! It's coming," the boy sat down and took hold of his left leg, the calf of which I could feel was in hard, tonic contraction, which state involved successively the left thigh, the left arm, the right arm, the right thigh and calf; the trunk was curved a little to the left, the boy's mouth was wide open and he continually exclaimed "Oh! oh!" as though in pain; relaxation occurred in about a minute and he then whimpered a little because of the pain he had felt. Consciousness had not been lost because he told what manipulation had been done to him during the cramp. This convinced us that we were not dealing with a true epilepsy. Dr. Burr requested a Wassermann of the spinal fluid, the first attempt without an anesthetic being interrupted by a severe seizure, and I felt fortunate in getting the needle out before it was broken. A second attempt with ethyl chlorid for general anesthesia was unsuccessful, as relaxation could not be obtained with as much of the anesthetic as was deemed safe to use; a third attempt with nitrous oxid gave alarming cyanosis without unconsciousness.

However, as increasing weakness of the grasp in the left hand and dragging with spasticity of the left leg made the diagnosis clear of irritation of the right motor area of the cerebral cortex, with the chances favoring a tumor rather than a gumma, because of the two negative Wassermann reactions, it was felt to be justifiable to explore this region by an osteoplastic resection, and the surgeon was accordingly called in consultation, who appointed a day of the following week for the operation. It was also deemed advisable to apply the old-time therapeutic test of a daily inunction of one dram of mercurial ointment. After the fifth inunction the nurse reported that the spells were quite mild and numbered only six in twenty-four hours; and on the day

set for the operation there were none, so surgical intervention was deferred. Thirty-five inunctions were given in the first course, followed by potassium iodid in increasing doses up to 15 grains three times a day. After three months another course of inunctions was ordered, followed by potassium iodid, the boy in the meantime having been taken to his father's home in the West.

June 10, 1918, he was brought again for examination with the report that he had missed school only one afternoon in the whole year. Measurements showed that he had gained 5 inches in height and 15 pounds in weight. The treatment as directed had been interrupted and the first seizure for over eight months occurred February 14, on going to bed. Inunctions were then given, as directed by the local physician, every second day, other attacks occurring on March 16 and May 20. Potassium iodid, 5 grains three times a day, was given from June 10 to June 21, with the idea of a "provocative" effect, when Dr. Kolmer took the blood again for a Wassermann with a +++ result. Inunctions were again resumed, over thirty being given, but a severe attack occurred early in the morning of July 29. Potassium iodid was next given and Kolmer and Schamberg were consulted as to the advisability of giving arsphenamin, but in view of the possibility of setting up a dangerous reaction in the central nervous system by the use of an arsenical preparation, it was deemed best to continue the older line of treatment. The next spell was on August 20, at 1:30 a. m., a very severe one, waking him up. He cried loudly during it, and said afterward that it helped to get his breath if he cried out. A prescription was now given for the mixed treatment:

Hydrargyri chloridi corrosivi.....	¼ grain
Potassii iodidi	2 drams
Syrupi sarsaparillae compositae.....	3 ounces

M. et Sig. One teaspoonful three times a day after meals, with water.

Two and five months later reports were made of no attacks, no word having been received now for six months.

This case illustrates well many of the problems of syphilis in children. First, with regard to the time of infection: The absence of hereditary stigmata (Hutchinson's teeth, scars around mouth and anus, keratitis, underdevelopment), the tardiness in the appearance of symptoms, the negative Wassermann of the father's blood, would point to an acquisition after birth, but these cannot positively exclude inheritance of the disease from the mother, who may have acquired post-conceptional syphilis (the father being uninfected) and may have transmitted it to the fetus in utero or to the boy shortly after birth, or even, as has been observed in some instances, several years later. The seven-year period in this case of apparent freedom from symptoms also cannot positively exclude a congenital origin, as such instances of syphilis hereditaria tarda have been reported.

The second feature to which the case report directs attention is the comparative rarity of symptoms of cerebral involvement in children, in spite of the fact, as Dr. Jeans will point out, that the cerebrospinal

fluid shows specific changes in a considerable proportion of cases (some investigators giving high figures). Dr. Graeme Mitchell has kindly consulted our records at the Children's Hospital, of the Wassermann tests made by the laboratory and gives the following report:

Total number of tests, 178. Negative results, 133. Positive results (including 4 doubtful negative), 46.

Age	Diagnosis	Reaction	Symptoms
2 mos.	Rachitis, bronchitis	Moderately positive	No cerebral symptoms
1¼ mos.	Congenital syphilis	Strongly positive	No cerebral symptoms
1½ mos.	Rhinitis	Strongly positive	No cerebral symptoms
1 mo.	Congenital syphilis	Strongly positive	No cerebral symptoms
5 mos.	Contraction of the iliopsoas	Strongly posuive	No cerebral symptoms
7 mos.	Bronchitis, dermatitis	Strongly positive	No cerebral symptoms
1½ mos.	Coryza	Strongly positive	No cerebral symptoms
2 yrs.	Cervical adenitis, congenital syphilis	Strongly positive	No cerebral symptoms
5 mos.	Cervical adenitis	Strongly positive	No cerebral symptoms
10 yrs.	Frontal sinusitis, bronchitis	Strongly positive	No cerebral symptoms
12 yrs.	Interstitial keratitis	Strongly positive	No cerebral symptoms
2 mos.	Congenital syphilis	Strongly positive	No cerebral symptoms
6 yrs.	Adenitis, cervical, axillary, inguinal	Strongly positive	No cerebral symptoms
1½ mos.	Epiphysitis, both femurs	Strongly positive	No cerebral symptoms
1½ mos.	Congenital syphilis, pneumonia	Strongly positive	No cerebral symptoms
1½ mos.	Congenital syphilis	Strongly positive	No cerebral symptoms
3 mos.	Regulation of feeding	Strongly positive	No cerebral symptoms
½ mo.	Congenital syphilis, stomatitis	Strongly	No cerebral symptoms
16 mos.	Congenital syphilis, acute gastro-enteritis, rachitis	Strongly positive positive	No cerebral symptoms
2 yrs.	Congenital syphilis, acute gastro-enteritis, stomatitis	Strongly positive	No cerebral symptoms
5 mos.	Congenital syphilis, erysipelas, enteritis	Strongly positive	No cerebral symptoms
22 mos.	Congenital syphilis, interstitial keratitis, chronic nephritis	Strongly positive	No cerebral symptoms. Motion in left leg sluggish
2 mos.	Congenital syphilis	Moderately positive	No cerebral symptoms
3 mos.	Regulation of feeding	Moderately positive	No cerebral symptoms
3 yrs.	Iritis	Moderately positive	No cerebral symptoms
5 yrs.	Chronic otitis media	Moderately positive	No cerebral symptoms
2¾ mos.	Gastro-intestinal indigestion	Delayed negative	No cerebral symptoms
4 yrs.	Multiple osteochondromata	Delayed negative	No cerebral symptoms
2 yrs.	Infantile palsy; flaccidity of both legs	Delayed negative	No cerebral symptoms
5 yrs.	Chronic interstitial nephritis, imbecility	Delayed negative	No cerebral symptoms
?	Food intoxication	Weakly positive	Convulsions after eating watermelon

Age	Diagnosis	Reaction	Symptoms
11 yrs.	Not made; seen only twice.....	Moderately positive	Bad headache; slight tremor of extremities
9 mos.	Convalescent anterior poliomyelitis	Weakly positive	For one month inability to hold head up; back muscles weak; no rigidity of neck; ankle clonus on left
9 yrs.	Mitral regurgitation, acute insanity?	Weakly positive	No cerebral symptoms
1¾ mos.	Gastro-enteritis.....	Weakly positive	Opisthotonus; conjugate deviation of head and eyes to right, internal strabismus, left eye. Died
4 yrs.	Spastic hemiplegia.....	Weakly positive	Sudden onset with convulsion; then weakness in left leg was noticed; on admission 2 weeks later; loss of power and sensation in left hand, K. J. +, suspicious Babinski, plantar reflex exaggerated, tongue protrudes to left, pupils equal; mind rational. Discharged 3 months later with spasticity of left arm and leg
9 mos.	Tuberculous meningitis.....	Strongly positive	Convulsions, general rigidity
5 mos.	Congenital syphilis.....	Strongly positive	Convulsions for 3 weeks after birth; retraction of neck and spasticity of arms and legs

The four last cases with the one reported make, in the forty-six positive reactions, five who showed symptoms suggestive of cerebral involvement, a little over 10 per cent., while changes in the cerebrospinal fluid might be expected in three times that number.

A review of the literature is not of much value at present because of its paucity since the introduction of the Wassermann test, but the test is tending to confirm the older clinical ideas on the subject which held the following conditions to be probably syphilitic: chronic hydrocephalus, not clearly following acute meningitis; hemiplegia, not following whooping-cough, chronic valvular lesions, or acute gastro-intestinal attacks with convulsions; local paralyses due to lesions in the nuclei or along the intracranial course of the cranial nerves, such as external ophthalmoplegia with ptosis and paralysis of the pupil, which are much more common in adults than in children, as is diabetes insipidus of syphilitic origin.

The diagnosis of the nature of the varied pathological lesions of the cerebrum in childhood must, of course, be subjected to a sharp revision in the light of the Wassermann test, but in estimating the value of this, care must be taken to avoid attaching too much importance to a negative result, especially of the blood. Comforting as a negative report is, it may, as in the case reported, lead to a postponement of necessary treat-

ment; but it is probable that a negative result with the cerebrospinal fluid is of more value than a negative one of the blood. Thus, in cases of idiocy, two of which have come recently under my notice, the tests of the spinal fluid have borne out the old clinical idea that syphilis is rarely productive of idiots; some neurologists claim that if the children of syphilitic parents survive infancy, they are more likely to be above than below the average in intellectual ability. Careful investigation of large numbers of idiots ought soon to settle this point.

PROGNOSIS

Our patient illustrates well the need for a guarded prognosis in cerebral syphilis, even in children who, as a class, react to treatment more promptly than do adults. The astoundingly brilliant result following the entrance into the case of our fleet-footed friend, mercury, filled up with hope that recovery would be complete and permanent, but considering the pathologic changes that undoubtedly existed in that patient's cerebral cortex, it is extremely likely that a certain amount of inalterable fibroid change is present which will probably give trouble, even if a "Wassermann cure" is ultimately obtained in the blood and spinal fluid. Could he be kept under observation until this result is secured, then, if the spells should continue to recur in their first type or become more epileptiform, as would be likely, surgical intervention would undoubtedly be justified to see if the fibroid area could not be removed without injury to the cortex.

CEREBROSPINAL INVOLVEMENT IN HEREDITARY SYPHILIS *

PHILIP C. JEANS, M.D.
ST. LOUIS

In this paper it is intended to show that contrary to most current opinion, involvement of the central nervous system in hereditary syphilis is relatively common, and that by statistical study there is no difference in the incidence of central nervous involvement in hereditary and in acquired syphilis. It is further emphasized that the treatment for neurosyphilis is the same and should be carried out just as intensively whether the infection is acquired or hereditary. As a matter of fact, the differences between hereditary and acquired syphilis are accounted for by the fact that in one case it occurs in a rapidly developing organism and in the other case in a mature organism.

There have appeared in the last few years a number of studies of the incidence of cerebrospinal involvement in acquired syphilis based on the examination of the cerebrospinal fluid. Most of these studies have been made on patients having syphilis it its early stages. There is practically nothing in the literature in regard to similar observations on patients with hereditary syphilis. There is a priori no good reason why hereditary and acquired syphilis should differ in this respect. In either case there is an excellent opportunity for the central nervous system to become involved during the general dissemination of the spirochetes over the body.

Neurologists commonly claim that not more than from 5 to 10 per cent. of individuals with acquired syphilis develop neurosyphilis. Estimates based entirely on clinical observation necessarily represent a minimum because they do not include those cases with vaguely defined clinical signs which we now know to be so frequently associated with abnormalities of the cerebrospinal fluid. Probably the largest and best studied series of cases is that of Mattauschek and Pilz[1] in

* From the Department of Pediatrics, Washington University School of Medicine.

1. Mattauschek and Pilz: Quoted by Fordyce, J. A. M. A. **71**:1023 (Sept. 28) 1918.

which 4,134 army officers were followed for from ten to thirty years. In that period 10.25 per cent. had developed paresis, tabes or cerebrospinal syphilis.

Since the era of laboratory diagnosis the various studies of the cerebrospinal fluid in acquired syphilis have generally shown a higher incidence of central nervous involvement than was found by clinical observation. There are certain difficulties with such diagnosis that need to be understood before such studies can be interpreted. A positive Wassermann reaction in the cerebrospinal fluid is not observed until the serum is positive, and even then it is usually delayed. Most of the recent laboratory studies include such early cases or are almost exclusively made up of them, thus making that most valuable laboratory test almost useless. At this early stage the diagnosis must be based largely on the increase in cells, globulin and albumen, and on the colloidal gold test. In many instances these changes are so conspicuous as to leave no doubt of the diagnosis, but there is always a large group of borderline cases in which the changes are inconclusive. Studies among individuals who have had syphilis for several years show a more clean cut differentiation between the positive and negative cases and give a better idea as to the proportion that will develop neurosyphilis.

Slight variations in the cerebrospinal fluid from the normal have been reported by some observers as occurring in the great majority of individuals with early acquired syphilis. These changes have been variously interpreted. Some have regarded them as being a part of a constitutional reaction to the general invasion of the spirochete and therefore in a large measure transitory. Others think that the central nervous system is invaded in the majority of instances and that in many individuals the infection later undergoes spontaneous resolution. Whatever the interpretation, it would seem that only the conspicuous changes in the cerebrospinal fluid have any importance as far as the welfare of the patient is concerned. When such changes are present one can scarcely avoid the conclusion that the central nervous system has been seriously invaded.

A review of the literature shows that in early acquired syphilis cerebrospinal fluid study reveals conspicuous changes in from 25 to 40 per cent. of the cases. The variations in the duration of the infection in these studies and the variation in the standards as to what constitutes conspicuous changes render it difficult to compile from these

statistics an average figure on the resulting large number of examinations. However, I think it would be fair to state that practically one third of all individuals with acquired syphilis in the early stages show changes in the cerebrospinal fluid which strongly indicate serious invasion of the central nervous system.

Later in the disease the Wassermann reaction on the cerebrospinal fluid plays a larger rôle in the diagnosis. In most instances there is either a complete fixation or a complete absence of fixation, thus making a clean cut differentiation. There are but few studies of the cerebrospinal fluid at this stage. Of sixty-four patients observed two years or more after infection, Engman[2] found 34.4 per cent. to have a positive Wassermann reaction in the cerebrospinal fluid and 76 per cent. of these showed clinical symptoms referable to the central nervous system.

Since about one third of all individuals with acquired syphilis show laboratory evidence of involvement of the central nervous system early in the disease and about the same proportion show more definite evidence of such involvement late in the disease, many syphilographers believe, and with justification, that those who show early evidence of central nervous system involvement, and perhaps only those are potential candidates for late neurosyphilitic manifestations.

With[3] and Ravaut[4] each report eight patients with hereditary syphilis examined in the first year of life with the finding of conspicuous cerebrospinal fluid changes in none. These infants are probably more fortunate than the next similar number would have been. However, in twenty-six patients with late hereditary syphilis With found definite pathology in eight, or 31 per cent. No other such statistical studies in hereditary syphilis have come to my attention.

For some time a spinal puncture has been made routinely on all children with syphilis coming to the Washington University Dispensary. Since this measure has been a part of the routine examination, seventy-eight infants under 2 years of age and 136 children over 2 years of age have been studied in regard to nervous system involvement. These patients have had much detailed study that is not evident in this presentation. They have been studied neurologically by Dr. Sidney I. Schwab and fundus examination of the eye has been routinely

2. Engman: J. A. M. A. **61**:735 (Sept. 6) 1913.
3. With: Brain **40**:403 1917.
4. Ravaut: Quoted by With,[3] Brain **40**:403, 1917.

made. Dr. John Green[5] has already reported the eye findings in more than 100 of these patients. It is intended to report these patients from a neurologic point of view at another time. The chief purpose of these statistics as here presented is to point out the frequency with which the central nervous system is seriously involved in hereditary syphilis.

It is usually conceded that if the cerebrospinal fluid gives a positive Wassermann reaction there is involvement of the central nervous system. The finding of increased cells, globulin, albumen and a positive colloidal gold reaction, though important, has not the same significance as a positive Wassermann. For this reason the Wassermann reaction is especially stressed in this study. The Wassermann reactions have been made by an expert technician using for each test 0.8 c.c. of spinal fluid and three antigens, simple alcoholic, cholesterinized and Noguchi's acetone insoluble lipoid antigen. By 4 plus is meant complete fixation with all antigens and by one plus is meant positive with the cholerterinized antigen only. The 2 plus and 3 plus are arbitrary gradations between these two extremes.

Of the seventy-eight infants the serum of all but one gave a strongly positive Wassermann reaction. The exception was an infant, 1 month old, who had a rash, rhinitis and infiltration of the palms and soles. The cerebrospinal fluid of thirty-one of these infants gave a positive Wassermann reaction. Of these, eleven reactions were 4 plus, one was 3 plus, ten were 2 plus and nine were 1 plus. Of the nine 1 plus reactions, eight were in infants under 3 months of age, i. e., at a time which might easily be too early for the Wassermann reaction to be strongly manifest even though the nervous system might be definitely involved. In six of these nine instances of 1 plus reaction the cerebrospinal fluid showed other changes corroborating the positive Wassermann reaction. Three were not otherwise examined, but in one a later specimen of cerebrospinal fluid gave a 4 plus Wassermann reaction.

Eleven of the thirty-one infants whose cerebrospinal fluids gave positive Wassermann reactions had clinical signs of neurosyphilis. The signs manifested were chiefly those of meningeal involvement with evidence of deeper involvement in addition in three. Three patients with convulsions were seen at the age of 3, 3 and 10 months, respectively, soon after the onset of the convulsions. One patient with neck rigidity and other signs of meningitis, but without convulsions, was

5. Green: Trans. Am. Ophthal. Soc., June, 1919.

seen soon after onset at the age of 7 months. Four patients with moderate grades of hydrocephalus were seen at the ages of 5½, 6, 9 and 11 months, respectively. Two patients with optic neuritis were seen at the ages of 1½ and 3 months, respectively. One patient with hemiplegia and hydrocephalus was seen at the age of 18 months.

Of the 136 syphilitic children from 2 to 14 years of age, seventy have been classed as having a clinically active infection, and sixty-six as having a latent infection. All but one gave a positive serum Wassermann. The exception was a child who had an acute dementing process and whose cerebrospinal fluid gave complete fixation. The children classed as having a latent infection were for the most part brothers and sisters of children having clinically active syphilis. Some of the children having latent syphilis had scars or similar signs of former lesions, but most of them had no signs or symptoms whatever except a positive Wassermann reaction.

Of the seventy actively syphilitic patients, twenty-two, or 31 per cent., had a positive cerebrospinal fluid Wassermann. Of the sixty-six latent infections, thirteen of the children, or 20 per cent., had positive cerebrospinal fluid Wassermann reaction. Of the total 136 older children, therefore, thirty-five, or 25.8 per cent. had positive cerebrospinal fluid Wassermann reactions. Of the thirty-five patients having positive cerebrospinal fluids, twenty-five had symptoms or signs referable to the central nervous system. In addition to the twenty-two patients with active syphilis, these twenty-five include three patients with fixed pupils who had no other signs of activity than a positive Wassermann reaction and who were, therefore, classed as latent. Of these twenty-five children having clinical signs of neurosyphilis, twelve had gross mental defect associated. The more superficial or meningeal manifestations occurred as follows: Two patients had acute meningitis with convulsions at 3 and 4½ years of age, respectively; one patient had meningitis without convulsions at 4 years of age; five patients had chronic convulsions which were classed as epilepsy (three of these had associated nervous manifestations); two patients had arrested hydrocephalus (one had associated nervous lesions). The submeningeal manifestations occurred as follows: six patients had hemiplegia at 2, 3, 3½, 4, 5 and 8 years of age, respectively; one patient had spastic quadriplegia at 3 years of age, and one had spastic paraplegia at 3 years of age; optic atrophy was seen five times, twice with associated lesions; fixed pupils without optic atrophy were seen four times;

one patient had an acute dementing process with the onset at the age of 6 years; one was seen when 11 years of age with juvenile paresis and two other patients had sensory changes with the cerebrospinal fluid reactions of paresis. Two of these latter three had optic atrophy.

It is seen, then, that the nervous system was involved in 40 per cent. of syphilitic infants, 31 per cent. of older children having active infection and 20 per cent. of older children having latent infection. Of the entire group of 214 infants and children the nervous system was involved in seventy, or 32.7 per cent. Of those having positive cerebrospinal fluids slightly more than one third of the infants and slightly more than two thirds of the older children had clinical manifestations of neurosyphilis at the time of observation.

Whether syphilis is hereditary or acquired, the earlier it is treated the better the prospect for complete cure. This is just as true for neurosyphilis as for infection elsewhere in the body. In the great majority of instances intravenous and intramuscular medication will result in the disappearance of the abnormal findings both in the cerebrospinal fluid and in the blood when such treatment is given early in the disease. Later in the disease these measures are often ineffectual, both clinically and serologically. Even the more drastic procedure of intraspinal therapy, though it may bring about a serologic cure, often leaves the clinical condition unimproved.

We have apparently succeeded in curing, both clinically and serologically, a large proportion of those children with early neurosyphilis, who continued under observation. Every one realizes how discouraging is the prospect of cure or even of benefit in children showing evidence of parenchymatous and in many instances of late meningovascular involvement of the central nervous system. In one sense we have succeeded in curing some of these children. We have treated them until the blood and cerebrospinal fluid are repeatedly negative, but we have not brought back the sight of a child with optic atrophy, nor in most instances have we restored function to a child with paralysis. The time to treat neurosyphilis is before it is clinically manifest. This can be done in at least two thirds of the instances of such involvement in infancy and in a considerable proportion at any age if we but take the trouble to examine the cerebrospinal fluid in any patient who has syphilis, whether there are any clinical indications for it or not. Justice to our syphilitic patients demands that the con-

dition of the central nervous system, as evidenced by the cerebrospinal fluid, be known before they are discharged as cured.

It is a common idea that hereditary neurosyphilis is a more hopeless state than if the infection were acquired and that it cannot be treated as intensively with safety. Such has not been our experience. Because of the fear of a Herxheimer reaction, we have been careful to have the patient well treated with mercury for a short period before starting arsphenamin, but after such a period it has been our custom to administer arsphenamin in full doses and at short intervals. In fact, the treatment of neurosyphilis has been more intensive than if such a lesion were not present, because the consequences of insufficient treatment are more serious. It has further been our custom to resort to intraspinous medication in those instances which did not respond with reasonable promptness to the other measures. We have nothing new to offer in the way of treatment except that we have demonstrated that children with neurosyphilis can be treated with safety by the same intensive methods that are used in other clinics for adults with acquired syphilis.

SUMMARY

According to a survey of the literature there is found involvement of the central nervous system, as shown by the cerebrospinal fluid, in about one third of all individuals with acquired syphilis.

Among the 214 patients of this study having hereditary syphilis, a similar incidence of involvement of the central nervous system has been found.

The treatment of hereditary neurosyphilis need differ in no way from that of acquired neurosyphilis, since it can be carried out safely with the same intensity in both instances.

DISCUSSION

DR. MARRIOTT: It is well to point out that these studies of Dr. Jeans' are of more than academic interest. The results such as he has obtained are of value in prognosis and treatment. Infants with positive findings in the cerebrospinal fluid are likely to develop serious symptoms following the administration of arsphenamin; this seems to be a Herxheimer reaction in the central nervous system. If, however, these children receive a preliminary course of treatment with mercury before the administration of arsphenamin such a reaction is less likely to occur.

Infants with involvement of the central nervous system are more difficult to cure than those who have not this involvement. The blood Wassermann may become negative and yet the Wassermann reaction on the spinal fluid

remain positive. This is an indication for further intensive treatment. Without spinal fluid examinations a certain number of infants would be discharged as cured when the disease is still active.

DR. DEBUYS: I should like to emphasize a few points. We should not be too eager to exclude congenital syphilis because of a negative blood Wassermann. We should always examine the spinal fluid. It is well to get away from the intraspinal method of treatment in congenital syphilis. That plan of treatment is being abandoned in adults. My attention was called several days ago to the work of two observers who showed the presence of arsenic in the spinal fluid a short time following the intravenous injection of salvarsan in adults, which would tend to demonstrate the permeability of the choroid plexus. This, however, does not conform to the observations made in the past by many observers.

DR. ABT: I would like to ask the manner of administration of the salvarsan and what preparation was used. I use both mercury and arsenic.

DR. JEANS: As to the preparation of arsenic, we are using arsenobenzol, standard dose, that is $\frac{1}{100}$ of a gram per kilogram of body weight. Of the mercury we use the routine injection of bichlorid, $\frac{1}{2}$ minim of a 1 per cent. solution per kilogram of body weight.

A HEALTH STUDY OF A BOYS' SCHOOL

RICHARD M. SMITH, M.D.
BOSTON

The health of schoolchildren has been a matter of interest to physicians and school authorities for many years. This interest has been given an added impetus as a result of the war and the general awakening to the importance of child life. Thus far the care of the health of schoolchildren has been for the most part along conventional lines. The primary effort has been directed toward the detection of physical defects and the listing of them. Notice has been sent to parents telling them of the need for treatment. In some instances treatment has been secured through the follow-up work of school nurses. Recently special school clinics, notably, dental clinics, have been established for correction of defects. This is especially true in communities where hospital facilities are inadequate. This development is a distinct advance over the work of previous years, but there is much still to be wished for in this direction. It has been customary also in some schools to provide lunches. For the last two years there has been unusual interest in malnutrition. This interest has resulted in a considerable measure from the weighing and measuring tests of young children instituted by the Children's Bureau. With the discovery of the prevalence of malnutrition has come a new emphasis on the necessity of proper food. Special classes have been formed for the feeding of underweight children.

In many schools the entire plan, as outlined above, for taking care of the health of the pupils has been in operation, and in nearly all schools some part of it. In only a few schools has there been any study of the health of the children taking into account all factors in the child's life and their relation to one another. The public schools have been more progressive than the private schools in this matter. In fact, many private schools, excellent in other respects, entirely neglect the question of health.

As pediatricians whose chief responsibility has to do with the health of children, there are certain matters concerning which I believe we should take definite action. These matters have to do with the quality of care given at present to the health of schoolchildren and

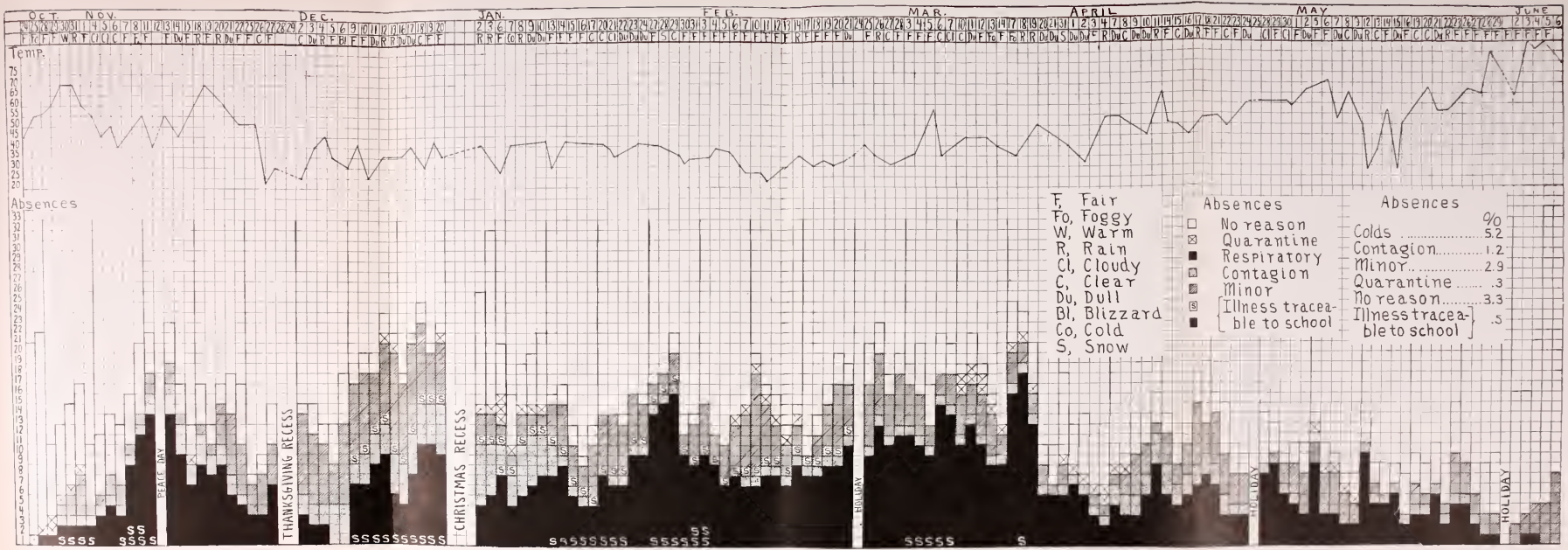
with further needed extension of care. There are certain minimum standards of health supervision to which every schoolchild is entitled.

In the first place, we should demand that every schoolchild be given a complete physical examination at least once a year, and means provided for the correction of defects and the treatment of diseases found. At the same time we should see to it that defects or diseases do not develop while the child is in school. Physical examinations should be made by a competent physician and made with the child undressed. I am told that at a recent meeting of a state medical society a well known school physician in a large city argued that it was impossible to undress schoolchildren for examination, and that we must content ourselves with the imperfect results obtained by unsatisfactory methods. I do not believe parents have any objection to an examination, provided they understand why the examination is made. Good medical advice cannot be given without careful examinations.

In the second place, we should insist on proper qualifications for school nurses. School nurses are really health instructors, and they must be selected because of efficiency in that work, not because of any other reason.

In the third place, we should bring about a better understanding and a closer cooperation between the school authorities and the family physician, and the practicing dentist. Often the advice given at school and that given at home are entirely different. The difference usually arises because of a failure of understanding. Unity of action must be secured for the good of the child's health. If minimum health standards for schoolchildren were established and taught, there would be no conflict of advice. This involves the introduction of the teaching of school hygiene in the medical schools — a development which is sure to come in time, and which I believe we should try to secure at once.

In the fourth place, we should continue our recent interest in the question of nutrition. We must recognize in this connection that the study and special care of undernourished children does not include the whole problem. It is not sufficient to weigh and measure all schoolchildren and then confine all our attention to those who prove to be underweight. This method gives no recognition to the equal importance of preventing malnutrition in those who at the present time may be of normal weight. It is to be borne in mind also that our standards


OCT.
NOV.
DEC.
JAN.
FEB.
MAR.
APRIL
MAY
JUNE
Temp.
Absences
THANKSGIVING RECESS
CHRISTMAS RECESS
HOLIDAY
HOLIDAY
HOLIDAY
F, Fair
Fo, Foggy
W, Warm
R, Rain
Cl, Cloudy
C, Clear
Du, Dull
Bl, Blizzard
Co, Cold
S, Snow
Absences
No reason
Quarantine
Respiratory
Contagion
Minor
Illness traceable to school
Absences
%
Colds 5.2
Contagion 1.2
Minor 2.9
Quarantine .3
No reason 3.3
Illness traceable to school .5

for weight and height are by no means positively established. It is quite possible that the true normal may be of wide variation. We must, therefore, see to it that all the children are in good nutritional condition. To this end school lunches of the right kind should be provided for all the children, and some supervision given to the methods in which the lunch is eaten.

In looking toward the future extension of health supervision there are certain additional features to which we must direct our attention.

First.—There must be a general recognition of the importance of health. The recent draft has shown that our present methods of safeguarding health do not produce satisfactory results. The army required healthy men as its requisite for service. Health is just as necessary for efficiency in peace as in war. This principle is fundamental for further progress.

Second.—There must be a realization among school authorities, both private and public, that the health of children is as important as scholarship, and that education must not go at the expense of health. In many individuals this will involve a complete change in point of view with reference to school problems, and in many instances a radical readjustment of the school program.

Third.—There is especial need of securing the cooperation of parents in protecting the health of their children. In general, parents have failed to realize the importance of health in connection with school. I believe this is due to lack of information. They are particularly careless in the planning of activities outside of school hours, such as special lessons and parties. These activities must be planned in connection with the school work, and not be added to that without reference to other matters. The child's life cannot be divided between school and home hours, and each planned independently, if good results are to be obtained. The school master and the parent, and those interested in child welfare, with the advice of the physician, must act together and see that the child's life as a whole is lived so as to protect and not injure his health.

Fourth.—It is important that the pupils themselves receive instruction regarding health. The teaching of health to pupils must be eventually in the hands of the school authorities. This can be done only when the school authorities recognize the importance of health and after they have received from physicians information as to the proper

things to teach. This makes it necessary for us to be ready to submit courses of instruction for teaching health to children. Thus far there has been very little thought given to this matter, either by physicians or by anyone else. We must consider it, however, in the immediate future if we wish to have the right things taught.

I have stated my conclusions first, because they seem to me of more value in that position. What I have to say with reference to the particular school is a description of the methods used there to safeguard the health of the children. I have had an opportunity of working with the head of the school in a practical demonstration of the principles which I have described.

Mr. Rivers' Open Air School for Boys in Boston was started in 1915. From the beginning health has been considered equal in importance to education.

There is an advisory medical committee to whom the details of the curriculum are submitted in order that education and health may be considered in relation to each other. The work of the school is conducted entirely in open air classrooms. The food is approved by the medical members of the advisory committee. The children have rest periods each day. This is a definite part of the curriculum, and adapted to the varying needs of the boys of different ages. This is particularly important with the younger boys, who cannot get the full benefit of school without the periods of rest. Each boy's school program is explained in detail to his parents and the importance of planning home time in relation to this program is emphasized. The parents are told that they cannot expect good results in scholarship or health unless they consider the life of the boy as a unit. The school sends the boy to his family physician for advice when the progress in school or the health of the boy is suffering from a physical condition needing correction.

There is a school matron, corresponding to the school nurse in the public school, whose sole duty is to look after the health of the children. She does this under the direct supervision of the head of the school with the advice of the medical committee.

Every pupil on admission to the school brings a medical chart filled out by his parents or by the family physician, a form of which is shown, giving the medical background of the boy. This is considered carefully in planning his school work, his rest periods, his diet and any

special supervision which is necessary. Each boy is given a complete physical examination at the opening of the school year. On the basis of this physical examination in conjunction with his medical history his school program is determined, and he is listed in one of several groups, either as "normal" or "for general observation," or as "overtire," i. e., a boy who has to be watched because of the possibility of fatigue, or as "colds," i. e., catches cold easily, etc.

The hours at school are divided into four groups, study, inactive, active and occupation. This is done in the belief that for each child there is a proper balance between his hours of study, activity and inactivity during each day. So far as the school is concerned, this can be carried out accurately. For its complete application it requires the willingness on the part of the parent to cooperate. Although the hours at school represent most of the boy's day, there is time before school and especially after school at night when there may be no supervision. If these out of school hours are given no thought, in connected with his school routine, his balance of time is disturbed. The number of hours of study for a boy of given age and grade is pretty constant and must remain so if he is to do the required amount of work and advance. The number of hours of inactivity, activity and occupation may be planned according to each boy's requirements. By periods of activity is meant school athletics and recess; by inactivity the rest period and dinner hour.

From a study of the boys in the school over a period of several years it has been determined that the following percentages of time represent about what the average boy of a given age can do without injury to his health and make satisfactory progress in his studies.

TABLE 1.—STANDARD PERCENTAGE OF TIME GIVEN TO STUDY, INACTIVITY AND ACTIVITY

Age	Study	Inactivity	Activity
6 years	4	3	2
7 years	5	2½	2
8 years	5	2	2½
9 years	5	2	2½
10 years	5	2	2½
11 years	5½	2	2½
12 years	5½	1½	2½
13 years	5½	1½	2½
14 years	5½	1½	3
15 years	5½	1½	3

It will be seen as the children grow older, the study hours increase slightly, the periods of rest decrease and the periods of activity increase. Each boy's schedule in percentages is checked against the formula for his age. This method has been of great value in many instances where boys were not doing well. This is not unlike the use which is made of caloric requirements in infants' feeding.

Every boy when he gets to school each morning is checked in on a chart by the matron. At this time inquiry is made as to his health. The matron also makes a superficial estimate of whether the boy looks well or not. If she is in doubt, the matter is pursued further. She sees that his clothes are right for the day. If a boy is not present and the reason is not known, the parents of the child are called on the telephone and the reason determined. The parent is asked, if the cause of absence is illness, where it was contracted. This is done in order that a record may be kept of whether the school was in any way responsible for the illness, i. e., whether it could have been prevented by any modification of the general school program or of that particular boy's program.

There is a general list of boys who need special attention which is posted in every school bungalow in order that the teacher may see and keep in mind the children in his class who require unusual care from the health point of view. If a boy on any particular day requires additional attention, the matron, at the time of checking in, makes a note of it. A list of these boys is taken to the teachers in charge of their rooms. Each boy when he leaves the school in the afternoon is checked out. At this time the matron sees that he has on enough sweaters to ride home; that he is not too hot or too cold or that his feet are not wet, and that he is physically in good condition. There is also noted in his checking out, if he leaves at any unusual time, the reason for it or any special comment. By this means of checking in and out, I feel confident that a great amount of good results. It makes a particular person responsible for the health of every boy each day. Between the time of checking in and checking out the matron spends her time in the schoolrooms, being sure that the boys who need special attention are receiving it. She sees that they are properly dressed for exercise and in general keeps track of the health program as outlined by the school.

A chart of absences is kept on which is recorded each day all the absences and the reasons in order that it may be visualized to compare

with other days. There is recorded on this chart also the daily temperature and the kind of weather. A study of this chart reveals several interesting things. There is a remarkably small amount of contagious disease. This, I believe, is due to the constant effort on the part of the school to exclude any possible sources of infection. Children are kept from school whenever there is known illness of this character at home. The chart shows a variation in respiratory diseases, primary colds, running with modification in the weather. It shows also the small number of cases of illness which can be traced in any way to the school. Table 2 is a summary of the chart.

TABLE 2.—ABSENCES

Total number of boys in the school at present, 115 (slight variation during year)		
Number of school days	142 (Oct. 25, 1918, to June 6, 1919)	
Possible total attendance	16,333 days	
Days lost through colds	859 days	5.2%
Days lost through contagion	197 days	1.2%
Days lost through minor illness	487 days	2.9%
Days lost through quarantine	51 days	0.3%
Days lost through no reason traceable to illness	541 days	3.3%
Days lost through illness traceable to school	94 days	0.5%

TABLE 3.—SCHOOL PROGRESS OF UNDERWEIGHT CHILDREN

Name	Weight Oct. 1918	Underweight for Height	Gain in Weight During Year	Scholarship	Days Absent	Usual Reason
B.	65 lbs. 13 oz.	5 lbs. 3 oz.	2 lbs. 11 oz.	Good	6	Colds
B.	83 lbs. 12 oz.	15 lbs. 4 oz.	9 lbs. 6 oz.	Good	12	Colds
C.	61 lbs. 5 oz.	10 lbs. 11 oz.	3 lbs. 4 oz.	Excellent	7	Colds
G.	53 lbs. 15 oz.	6 lbs. 1 oz.	2 lbs. 3 oz.	Good	12	Colds
K.	56 lbs.	6 lbs.	1 lb. 2 oz.	Excellent	25	Colds and chickenpox
P.	67 lbs. 12 oz.	7 lbs. 4 oz.	2 lbs. 14 oz.	Excellent	6	Colds
P.	65 lbs. 8 oz.	11 lbs. 8 oz.	4 lbs. 4 oz.	Good	6	Minor
McP.	75 lbs. 3 oz.	24 lbs. 13 oz.	8 lbs. 5 oz.	Excellent	2	Minor
P.	88 lbs. 10 oz.	18 lbs. 6 oz.	9 lbs. 10 oz.	Excellent	6	Minor
W.	58 lbs.	4 lbs.	2 lbs. 10 oz.	Excellent	3	Minor
W	58 lbs. 10 oz.	3 lbs. 6 oz.	2 lbs. 3 oz.	Good	5	Chickenpox

There is a table of some of the boys who were below par on entrance, as indicated by being underweight. The progress during the year, the gain in weight, the scholarship record, the number of days absent and the usual reason are also shown. It will be seen that many of the boys made good progress in scholarship but had colds, and that others made good progress despite the fact that they did not make satisfactory gains in weight. A similar table might be shown of boys who did not make good progress in scholarship or who had many

absences due to illness, but who had a normal weight at entrance and made good gain in weight during the year. Some of these boys had a physical handicap accounting for their poor scholarship, which was corrected, and then they did well in school work. These boys would have been neglected in health and in scholarship if the weight only had been considered as an index of health.

It may be worth while to cite the record of a few boys.

CASE 1.—B., 12 years of age; normal weight for height. Had a history of repeated colds. Physical examination was negative except for a rather "ragged" throat. His progress in school was slow. He was absent often. Colds were cured by medical treatment, and then progress in school was good and he passed from the lower end of the grade to a candidacy for first position. His attendance was normal.

CASE 2.—D., 9 years of age; was underweight for his height (11 per cent.), and in poor general condition. Physical examination was negative except for the undernourishment. He did poor work at school and had many absences. A conference was held with his parents and the physician and it was found that after school at home he overexercised with resultant fatigue and interrupted sleep. His hours out of school were regulated in connection with the school schedule. The result was a marked improvement in school. His health is excellent, although he made only slight gain in weight (1¾ pounds).

CASE 3.—H., 12 years of age, underweight (12 per cent.), with normal past history, except that he was "never robust." Physical examination negative except for the undernourishment. His scholarship was very bad. Absences were frequent. His schedule was arranged with special attention and more rest, and more supervision at meals to insure his taking the proper amount of food. The result was that his scholarship became excellent. His general condition was markedly improved. His weight is within a pound and a half of normal.

CASE 4.—F., 13 years of age; underweight (10 per cent.); gave a history of repeated colds. Physical examination was negative except for chronic infection of the tonsils with enlarged cervical glands. He did poor work at school and was absent repeatedly for tonsillitis. His tonsils were removed. His progress in school became excellent. He had no more absences and gained to within one and a half pounds of normal weight.

CASE 5.—P., 12 years of age; underweight (24 per cent.); had a history of repeated absences from school. He never felt well and made very poor progress in the classroom. Physical examination was negative, except for the marked undernourishment. It was quite evident that the difficulty with this boy was malnutrition and that he was constantly overtired. His schedule was made out with this in mind. By Christmas he began to show the effects of the special care. During the second half year he had no absences, and he is now one of the best students in the school. His gain in weight was only fair. He is now 16 per cent. underweight. Special watching by the matron made the change in this boy.

It is difficult to compare results from the health standpoint in this school with those in other schools, because of the lack of uniform standards of estimate. I am convinced, however, that progress in school health supervision must be in general along these lines: first, recognition by school authorities of the importance of health and the necessity of arranging school work with this in mind, and second, cooperation of parents and those interested in children in planning the time out of school in relation to the school program.

329 Beacon Street.

DISCUSSION

DR. TALBOT: I think this paper of Dr. Smith's is very important because Dr. Smith has had an experimental laboratory in which he has tried out what physicians think should be done in schools, and so far as I know it is the first time that everything has been done which the physicians want. Dr. Smith has brought out or will bring out in this publication a very important element of health regulated by physicians. It furthermore emphasizes the factor of undernutrition; some of the children being 10 or 12 per cent. underweight. But underweight is not the whole thing. It emphasizes also that the basis of undernutrition studies is not a sound one. Dr. Holt made a step in the right direction when he studied the weights and heights of children in a private school. The great difficulty we have had to meet in our metabolic studies is, What is a normal child? There is no criterion by which to go. The most important thing is to determine what is the normal child. The emphasis which the doctor has laid on the individual factor in dealing with health has been too long neglected. Dr. Smith has opened up a subject which is going to go a long distance and which is of great importance.

DR. HAMILL: I want to congratulate Dr. Smith on being among the first to present a rational plan for the medical supervision of the schoolchild. Medical supervision has been neglected in the country day school without doubt, but I have been dumfounded at the inadequate supervision of the health of the boys in our large boarding schools. Dr. Smith's paper will be of great help in awakening the school authorities from their present indifferent attitude toward this subject.

There should be made on admission to the school not only an examination for the recognition of physical deformities and defects but the condition of nutrition of the boy should be carefully studied and noted and a full study of the boy's previous health and habits should be made in conjunction with the parents.

You are all familiar doubtless with the studies of Gerhart of the food intake of the boys in one of the largest of the New England schools. These studies have not received the attention that they should have and they must not be passed by without criticism. They showed that the boys were taking on an average 5,000 calories per boy per diem. If that was the average, the food intake of some of them must have been absolutely magnificent. The interpretation of the results of these studies was most unsatisfactory. The dietary of the school received the endorsement of the investigator. Any trained physician facing an audience of boys in any of our boarding schools will quickly pick out a large percentage of boys whose condition of nutrition is abnormal.

It is not only the boy that is underweight that requires attention, but the boy who is overweight as well. We have all seen the large, fat, flabby, indolent and mentally sluggish boy whose food intake is beyond the power of any human organism to handle. We have also seen the marvelous transformation brought about in such a boy, especially in his mental condition, when his diet has been restricted and his general habits of life have been reformed.

It is most unfortunate that a dietary for a boys' school averaging approximately 5,000 calories per boy per diem should be endorsed as satisfactory. At least there should have accompanied such a statement the supportive evidence of a careful study of the condition of nutrition of each boy, made by some one in whose judgment the medical profession could have placed implicit faith.

Dr. Smith is to be congratulated on having brought this subject to our attention in this interesting form. It is one that must be worked out and it is not the function of the physiologist alone but in conjunction with the pediatrist.

DR. SOUTHWORTH: I am interested in the extension of this work in an advisory capacity to the home. A good many teachers limit their observation to the school and seem to fear to encroach on the family physician's ground in the management of the child in the home. I have in mind one boy, not under my care, who has not done well in one school which he attended, but did much better when transferred to another. His was a peculiar temperament of the perpetual motion type and he did badly in school. He was always in mischief. I happen to know something about the home life. He had French at breakfast and French or another language at lunch. He also had music lessons. He was penalized by being denied sports if he did not do well in school. On holidays he was taken on some instructive expedition. The boy was already of an extremely nervous type and was being constantly pushed at home so that he had no chance for mental recuperation. If he had had the right sort of oversight together with conferences with his parents, it would have improved his health as well as his mental standard. I think this explains in part why children sometimes do better in another school, where their routine is arranged differently.

DR. LA FÉTRA: I want to congratulate Dr. Smith on having arranged regular periods of activity and inactivity for individual boys. Many pupils who do poorly do so because of lack of consideration of the three factors Dr. Smith has mentioned. One factor which is often lost sight of, but which should be emphasized, is the period of rest and repose between study periods. Efficiency engineers have found in their study of the factors producing fatigue that a greater amount of work and more accurate work can be accomplished by the introduction of short rest periods. The degree of fatigue, the number of absences, and the labor turn-over have been greatly decreased by the introduction of five or ten minute rest periods every forty-five or fifty minutes. Of course the same principle should be applied to the growing child.

Another factor which is of great importance, not so much in the private as in the public school, is that the length of time allowed for lunch is altogether insufficient. The child does not have time to chew his food. Of course individuals are different; but the ambitious child who wants to do well in school is in a state of nervous tension when the lunch period is short and he eats his meal with anxiety. In the public schools throughout the country there should be sufficient time allowed for the children to eat the meal without hurry and the period should be long enough to allow for a short rest before going back to the classroom.

DR. ABT: If teachers knew more about health and doctors knew more about education we should have better results. Many doctors go into the work unprepared. The work the doctors perform in many cases (I am not casting any reflections on anybody) is perfunctory and done in a haphazard way. We need full time men who will devote all their time to this work. There is no way for the men to learn this work. They should be taught hygiene before they leave the medical school or before they go into the public schools as medical inspectors. I think this matter of school inspection has a brilliant future but at the present time it is a failure. The matter of school inspection should be left to the best men in the community and not to the young men who know little or nothing about the work.

DR. HELMHOLZ: I would like to emphasize this matter from another point of view. If one looks over our program he will find that there is but one paper which has to deal with preventive medicine. As pediatrists, we as an organization have strangely neglected this phase of our work and it seems to me we should give more attention to preventive pediatrics. Once every year or second year we should devote a session to the discussion of preventive medicine as it is related to pediatrics.

DR. SHAW: I want to emphasize Dr. Helmholz' criticism of the present method of medical school inspection. One reason why it is a failure is the indifference and lack of knowledge on the subject by the pediatricians. The term medical school inspection is a poor one and is being superseded in some cities by "medical school health supervision." The medical colleges are sending out young doctors with little or no training in social preventive pediatrics, which includes school health work, problems of infant mortality, nutrition, etc. New York state is about to lose one of its foremost educators, Dr. Finnegan, who is going to direct the educational policy of Pennsylvania. He believes that health should occupy an important place in the school curriculum, and that schoolchildren should pass examinations in health as well as the three R's. He has no faith in the present method of medical school inspection which collects statistics and is of no practical value.

DR. HOLT: We all realize the difficulties and the defects in the present situation. It is the remedy that is to be considered. The hours for schoolchildren are in most communities the same as they were thirty or forty years ago. The necessary changes can be brought about only by educating public opinion, and that is certainly one of the functions of this society. It is not only possible but absolutely necessary that there should be a radical revision of the work being done in our schools, both public and private.

DR. HEIMAN: This excellent work is not only good for school life but should be extended to camp life. I know that in some camps children are overexercised and have little medical supervision. I think summer camps should adopt this method.

DR. SMITH: I do not want to give the false impression that the parents of every child cooperate with us. In many instances we have succeeded in convincing parents of the advantage of giving their cooperation.

The physician and the teacher often look at the child from different angles. It is essential that they come together and talk matters over, if the best results are to be obtained. It is necessary also for the parents to be interested in the health of their children. They are too frequently concerned only with the marks which the child gets and pay little attention to the effect the school may have on health.

In connection with what Dr. Southworth said about lessons outside of school, I would like to say that I think these things ought to be planned in connection with the school work. If a boy rides horseback on Friday afternoon, for instance, this must be recognized as a part of his exercise for that day and his school program must be planned accordingly.

May I call your attention to one other matter which I neglected to speak about? You will see that the majority of the absences in school are due to illness. If teachers desire a good attendance, it is necessary for them to consider the question of health.

DR. HAMILL: Has there been any improvement in the scholarship of the students since the operation of this health program?

DR. SMITH: It is difficult to answer Dr. Hamill's question since the school has always been under this regime. There are a few boys, however, who are doing well now, but who had difficulty in other schools. Not every boy has made a success in the school. The reason for this is because of the inability to secure the parents' cooperation and also because the time outside of school is not arranged in connection with the school program.

THE PREDOMINANCE OF SEBORRHOEIC ECZEMA IN EARLY LIFE

THOMAS S. SOUTHWORTH, M.D.
NEW YORK

At the present moment, our views concerning the large group of cases formerly classed as eczemas are undergoing revision. The similarity of early forms has led to a wider application of the term dermatitis, especially in the acuter types. There is also a further tendency to separate those cases in which there is a demonstrable sensitization of the body to proteins, and to make a distinction between eczemas of external and internal origin.

The recent trend of pediatric literature appears to be that of considering the majority of infantile eczemas to be due to dietetic or other internal causes, and the value of the excellent work done in this direction, both from an etiologic and a therapeutic standpoint, is fully acknowledged by the writer. Nevertheless it would appear that the part played by seborrhea has been overlooked and has failed to receive the recognition which it deserves.

In our pediatric literature and textbooks, eczema is described without any reference to seborrhea or else the latter is dismissed with brief mention as an entity apart. Kerley indeed mentions certain intertrigos which responded to treatment on this basis, but nowhere is there any suggestion that any considerable part of eczemas in early life have this foundation.

My own observation, on the contrary, has been that a preponderance of the cases of eczema presenting themselves in the ordinary routine of pediatric practice are associated with seborrhea, many of which are mild and would rarely reach the dermatological specialist. The prevalence of milder types led me to examine the severer eczemas with a view to determining the presence of seborrheic manifestations, and I have not been surprised to find them frequently present.

The clue to such basic causation is found about the ears as well as upon the scalp. To trace the seborrheic factor it is necessary to begin with the milder forms upon the face and body whose identity may be established by finding seborrhea upon the scalp and in the characteristic location in the folds above and below the ear. In such cases, if the present condition of the scalp does not permit of an exact

diagnosis, inquiry will usually elicit the information that the "milk crust or cradle cap" persisted unusually long and was eliminated with some difficulty.

Seborrhea of the scalp it is unnecessary to describe, but the ear signs may vary from a mere adherent dry exfoliation to a moist and exuding surface where two surfaces are in contact, either above where the pinna joins the hairy scalp or below at the juncture of the lobe of the ear with the upper part of the neck. When marked, and with some severe eczemas, the moist surface may invade considerable portions of the sulcus behind the ear.

There is something significant also in the predilection of such eczemas for fat babies fed on breast milks rich in fat and for bottle babies suffering from disordered digestion caused by a relatively excessive intake of fat. In both groups of infants we might expect the fat glands of the skin to be more readily subjected to seborrheic disturbance. Towle and Talbot, in their description of cases of eczema associated with faulty digestion of fat, noted "the tendency of the exudative eruption to remain limited to the regions of the head for an appreciable length of time, even for months. Many cases, in fact, showed no tendency to spread to other parts."

Many must have been struck by this proneness to localization, for which no explanation has been forthcoming other than that the eruption tends to appear upon the blushing areas where presumably there is greater vasomotor activity. Such an explanation is not of much etiologic value.

Neither does any internal cause serve to explain this limited and peculiar localization, since any sensitization to proteins or toxins which have gained access to the body should give rise to generalized manifestations, unless, indeed, there be some local predisposition to determine its localization.

That such a predisposition exists when there is visible seborrhea in the neighborhood, would not appear to be a presumptious assumption. In nearly every case of marked facial eczema some characteristic lesion will be found, if sought for, above or below the ear. No attention has heretofore been called to this association, and it has been generally overlooked. When the auricles are massively involved in the eczematous process, the characteristic lesion is, of course, overshadowed.

Some explanation also is due us for the fact that such facial eczemas tend to involve the hairy scalp rather than the neck and chest. Here

the acceptance of a seborrheic predisposition appears particularly reasonable.

While McKee has wisely said that the diagnosis of an internal cause for eczema should not be made until all external causes have been eliminated, there is no real conflict between the two since internal causes undoubtedly predispose the skin to succumb to external factors. Indeed it is, or should be, recognized that the seborrheic sicca of adults suffers exacerbations with a lowered tone of the body or faulty assimilation due to excessive intake of certain types of food.

Although Pusey states that seborrheic dermatitis of itself does not become of sufficient intensity to produce a weeping dermatitis, we need not limit its possibilities to the dry scaly type, to which our understanding of seborrhea is so commonly restricted. He himself admits the possibility of infantile eczemas of the face and scalp being manifestations of a seborrheic dermatitis, and says that seborrhea of the axilla may become macerated, just as I have above pointed out that in infants it may become moist behind the ear.

To grasp the possibilities of seborrhea in furnishing a starting point for eczema, we must realize the ease with which other inflammations and infections are engrafted upon a seborrheic neighborhood. I have seen an intense dermatitis produced about the seborrheic ear of an adult by the application of a solution of carbolic acid purchased at a drug store. Here the preexisting seborrhea undoubtedly constituted the predisposing cause for so violent a dermatitis.

The recognition of a seborrheic basis for a lesion by no means excludes the possibility of other micrococcic invasion. On the contrary, it is probable that a majority of the more typical facial eczemas of infancy are associated with a mixed infection.

If further criteria be necessary, such may be found in the recalcitrancy of this group of eczemas to bland ointments. Oxid of zinc ointment, so helpful in other types, is, as a rule, useless in seborrheic cases of even the simplest form. Something else is necessary.

In this connection, we note the prevailing use of the term "stimulating" to denote such remedies as mercury, tar and resorcin, commonly employed in various forms of eczema, including the seborrheic. This term undoubtedly arose from the efficacy in some of the chronic dry and thickened types where stimulation is necessary.

Such stimulation would not only be unnecessary but possibly harmful in the more acute and moist types. Yet since we find them distinctly

helpful in the moist intertrigoes of seborrheica, as well as in the dry ones, it is probable that their efficacy is due to their germicidal properties. Indeed, it has been my experience that the bland and soothing ointments of the zinc oxid sort are unavailing until to them are added ammoniated mercury or tar, or resorcin to destroy the micro-organisms present.

Beyond pointing out this fact, I shall not go further into the treatment. We all recognize the part played by diet, both in the causation and the cure of the severe facial eczemas of infancy, and that this dietetic factor must be differentiated and corrected before the larger number will yield to our efforts.

My object is attained if I have called attention to the number of minor cases of dry seborrheic eczema, and to the frequency with which scalp and ear manifestations of this condition accompany the aggravated types of facial eczema in infancy.

807 Madison Avenue.

CHILD WELFARE

REPORT FROM INTERNATIONAL CONGRESS

FRITZ B. TALBOT, M.D.,
BOSTON.

Child welfare is an essential part of a general program in public health and preventive medicine and cannot be separated from it. It is particularly closely associated with housing and food production and supply, for children require food not merely for sustenance but for growth.

Children, as the weakest members of the community, feel first and most heavily the effects of all unfavorable hygienic, social and economic conditions. The war has increased infant and child mortality, and in some countries the birth rate has been lowered more than half. The health of many children who have survived has become greatly impaired by reason of improper food and neglect. Furthermore, the war and recent epidemics have enormously increased the number of dependent children, especially in the devastated countries.

A reduced birth rate and an abnormal sickness and death rate may be expected to continue for several years to come; a reduced birth rate, owing to death of potential fathers, to social and economic conditions which have affected the health of potential mothers, and to many other factors; an increased sickness and death rate, because of the scarcity and high cost of food, and general hardship and privation which favors the spread of epidemics and lowers resistance to all forms of disease. A world wide child welfare campaign is, therefore, one of the most urgent needs of the day, both from a humanitarian and economic standpoint; as the children are the nation's greatest asset.

The pressing problems now are:

1. To save the infants yet to be born and to promote their healthy development.

2. To restore the health and make possible the normal growth and development of children who are now suffering from disease or defective nutriment and to safeguard the health of those whose nutrition has not yet suffered.

3. To do something for the immediate needs of dependent children.

Experience has shown that no efforts in public health work produce more immediate or far reaching results than those which are put forth in child welfare.

The work relating to child welfare may be divided into six parts, corresponding to the period of child life to which it is directed:

First.—Eugenic consideration affecting the prospective parents.

Second.—The prenatal period in which the child is saved through care for the mother.

The importance of this may be appreciated by the fact that even in the highly civilized countries in peace times

(a) The still-births are equal to between 4 and 5 per cent. of the living births.

(b) That as a result of premature birth many infants born die in the first weeks of life.

(c) That other causes of the high mortality during the early weeks are due to conditions affecting the mother.

Methods of prenatal care will necessarily differ in urban and rural communities and also be modified by race, customs and environment. The essential features must be the supervision and education of the expectant mother by trained public health nurse, midwife, or health visitor from an early period of her pregnancy, aided when possible by the advice and assistance of a physician. The work is greatly facilitated by voluntary registration of pregnant women, which should be encouraged everywhere, and it should be connected closely with maternity and child welfare centers.

Success in any effort in child welfare is largely dependent on the intelligent cooperation of both parents; no plan of education, therefore, is complete which does not include the father. This is particularly important in the prenatal period and in the period of infancy.

Third.—Obstetrical care, which should include the services of a nurse as well as those of a physician or midwife. Proper care saves the child from accidents during delivery; saves the mother from immediate and remote dangers incident to her confinement; greatly increases the chances of successful breast feeding and prevents much blindness.

Fourth.—Birth to school age. This period is sometimes divided into infancy — including the first year or possibly two years — and a preschool age, extending from this time up to the fifth or sometimes the sixth year.

The important concern during the first two years is the child's nutrition; and the essential thing is that the child be kept under continuous observation and supervision. Everything possible should be done to promote breast feeding, as by far the most important means of insuring vigorous growth and development and preventing infant mortality. At first, the supervision must be close, the infant being seen in many cases every week or even oftener. The visits will be less frequent as the child grows older, being made at least monthly after six months, and every two months up to the end of the second year. Success during this period depends almost entirely upon our ability to educate the mother and to arouse and maintain her interest in her child's growth and progress.

The principal methods are:

1. Group instructions and individual advice in a central place known as a "milk station," a child welfare station, a "consultation," etc., and

2. Home visiting by public health nurses or health visitors.

Both methods have their place and should usually be combined, certainly in urban communities.

The problem of infancy up to the school period is much the same and can be carried on with the same organization — a quarterly visit to the home or by the child to the central stations and a full medical examination once a year should be the minimum. Much better results would be obtained if the observations were made more frequently. The above requirements should be for every healthy child. Those who are delicate or ill should have special medical attention.

By these means the beginning of organic disease may be detected, physical defects and deformities prevented or corrected, and a general supervision maintained over the child's diet and general hygiene.

Records should be kept of all abnormal conditions and any serious illness, also of height and weight, which will in themselves furnish a good guide as to health progress.

Permanent institutional care for infants and young children should be discouraged on account of the almost insuperable difficulties in maintaining nutrition in infancy under these conditions, and because of the great susceptibility of young children to infection, preference should be given to placing such children in suitable families. All creches, day nurseries, and the like should be under close medical attention.

Fifth.—In the School Period. From the sixth to the fourteenth or sixteenth year, the problem is to secure normal growth and physical and moral development, to recognize and correct defects which interfere with these, and to reduce to the minimum the risks of contagious disease. The school physician, the school nurse and the school teacher all have important functions. The school, furthermore, offers an opportunity, the value of which is only beginning to be appreciated, to interest, instruct and train the child himself in health matters. It should be the duty of schools to teach health as much as to teach reading and writing. Health work for the school children is inextricably bound up with the problems of general education, and all teachers should receive training which would qualify them to teach the simple facts of health and personal hygiene.

An important kind of health education which can be carried on in the school is the instruction of girls from the age of 10 years upward in the rudiments of infant care. This has been proved to be valuable in preparing them to undertake the responsible charge of their younger brothers and sisters, and what is even more important, in assisting to prepare them for the future duties of motherhood.

The child's mind is virgin soil for health instruction of every sort. It is not necessary at this period to clear away the rubbish of prejudice, superstition and habits based upon ignorance, which make health education so difficult and often so futile in adult life. Scientific facts in regard to food, health habits, hygiene and the spread and control of disease can, if presented by writers and artists who know how to reach the child's mind, be made both interesting and attractive. Why should not the stories of the conquest of smallpox, malaria and yellow fever be made as thrilling as the conquests of Alexander or Napoleon?

This instruction should be begun with children of eight or nine, for early impressions are the most lasting. The bare facts of hygiene or physiology as presented in school textbooks make little impression that is permanent. Their acquisition is made work. Health instruction should and can be made play. Health may even be made a competitive game. If the school children can be properly instructed in health matters we need have no anxiety about the adults of the next generation.

Progress in the pupil's health is best shown by the weight chart; thus an incentive can be furnished for the observance of health rules which are taught.

Health records, at least a record of weight and growth, should be kept during school life. In an ideal scheme, a medical examination should be made once a year of every school child, and those who are found to be physically below normal or who do not make a normal gain in weight should receive special attention and supervision during their school life.

Sixth.—Industrial period from the fourteenth to the eighteenth year. In connection with legislation relating to child labor, the following fundamental principles should be embodied:

(a) No child should be allowed to enter industry without first passing a physical examination by a competent physician, showing that he is able to perform the work intended.

(b) No child should be permitted to continue in any form of work which prevents his normal growth and physical development as determined by accepted standards, and periodic examinations made up to the age of 18 years.

Any intelligent action must be based upon an exact knowledge of conditions. Therefore, the first step should be a survey.

After a survey has been made in any activities which are contemplated there are certain fundamental principles which should be kept in mind if the work is to be effective and anything permanent accomplished.

(1) Local customs, climatic conditions, diet and other things on which are based the habits of the people should be carefully considered and appreciated.

(2) All local health habits and racial customs, which are good, should be accepted and used as a foundation on which to build.

(3) It is impossible to formulate beforehand a plan of procedure which can be followed closely. Any plan adopted must have great elasticity and be modified by circumstances as they arise in the course of the work.

(4) Local agencies, organizations and individual workers should be developed and utilized so far as possible.

(5) The utmost tact is necessary to adopt knowledge and experience gained elsewhere to work which is instituted under new conditions and among new people.

(6) No plan of work should be made so complex or so expensive that it cannot be duplicated or extended by a simple organization elsewhere.

(7) Success depends much less on the program adopted than on the personnel which will put it into effect.

(8) To carry out effectively any scheme of child welfare such as has been outlined, it will be necessary to provide material assistance in a considerable proportion of cases.

Dr. Lucas presented an additional detailed program for child welfare work in the community. It was recorded with the above program.

The Council at Cannes adopted, among its final resolutions:

(1) That the control of typhus fever be at once undertaken as an emergency relief measure.

(2) That the wide extension and development of child welfare work be selected as the first important constructive activity.

(3) That the training of schoolchildren in all grades by thoroughly qualified teachers in the subject of personal and general hygiene and the inculcation of proper health habits during school life are perhaps the most important undeveloped measures for permanently improving the health and contributing to the welfare of the people.

Finally, a communication from the acting secretary of the League of Red Cross Societies, dated June 2, 1919, from Geneva, Switzerland, states that "It is now confidently believed that the League of Red Cross Societies, now prepared to engage in a concerted and intelligent effort in behalf of the health and welfare of the world, will prove to be one of the most significant of modern institutions for the advancement of civilization. The series of recommendations will form the foundations for the practical work of amelioration which the League of Red Cross Societies is about to undertake on a world embracing scale.

ANEURYSM OF THE ASCENDING ARCH OF THE AORTA IN A BOY OF THIRTEEN

HENRY HEIMAN, M.D.
NEW YORK

This case is presented because of its infrequent occurrence and the interesting roentgen-ray findings.

The patient, a boy of 13, was admitted to the pediatric service of Mount Sinai Hospital about three months ago. The family history records the death of the father from epilepsy nine months ago. The mother had two miscarriages; otherwise negative.

In the past history there were several attacks of acute articular rheumatism, the last one three years ago. With the exception of measles, seven years previously, there were no other illnesses.

The present history dates back eight years with a gradual onset of dyspnea on exertion, precordial pain and slight cough which have grown progressively more marked. Recently he had been at the Montefiore Home and returned in fair condition.

On physical examination, the interesting signs were found in the cardiac and liver regions. On inspection, there was a marked pulsation of the carotids and the apex impulse was seen considerably to the left of the midclavicular line. There was no bulging at any point, no thrill to be felt, or any expansile pulsation. No tracheal tug was present. Percussion showed the heart enlarged especially to the left, the apex was 11 cm. from right border of sternum. There was rather a wide area of dulness at the level of the first and second interspaces, extending to the right 6.5 cm. and to the left 4.5 cm. from the midsternal line. On auscultation there was a systolic murmur best heard in the third right interspace and extending downward and upward. There was no bruit and the pulse was Corrigan in type, and capillary pulsation was present. Pulses were equal. The blood pressure in each arm recorded as varying only 5 mm., 140 and 145 systolic and diastolic. The polygraph tracing showed nothing unusual. The electrocardiogram showed marked left ventricular predominance, notching of R in Lead II, inversion of T in all three leads. Fluoroscopic examination was especially interesting, revealing an aneurysm of the ascending arch of the aorta.

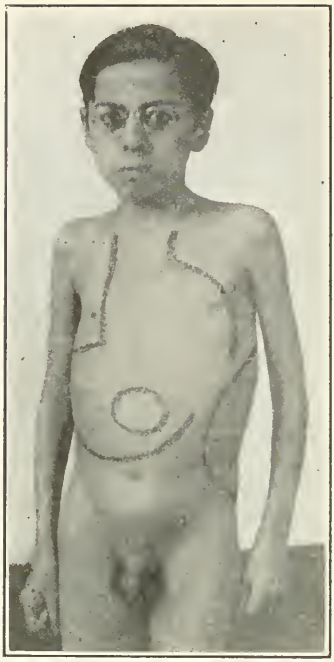


Fig. 1.—Patient, boy 13, showing increased area of cardiac dulness. Note position of hard epigastric mass, probably a gumma. Spleen is markedly enlarged, extending to the iliac crest.

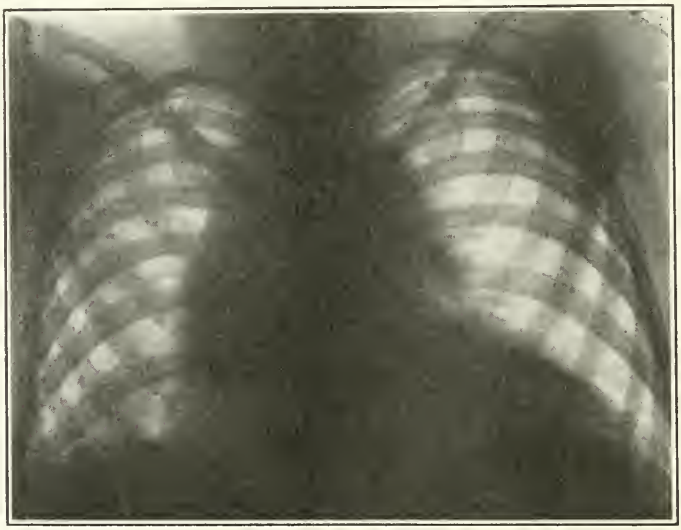


Fig. 2.—Roentgenogram of chest showing enlarged heart with apex 11 cm. from right border of sternum, with marked enlargement to right and left of sternum in the first and second interspaces, indicative of an aneurysm of the ascending arch of the aorta.

The roentgen ray corroborated this finding. The Wassermann test was ++++. The mother's Wassermann also showed a ++++ reaction. In the epigastrium there was a mass hard, rounded about 6 cm. in diameter, extending down about 8 cm. from the ensiform, movable, not tender, probably a gumma. The spleen was markedly

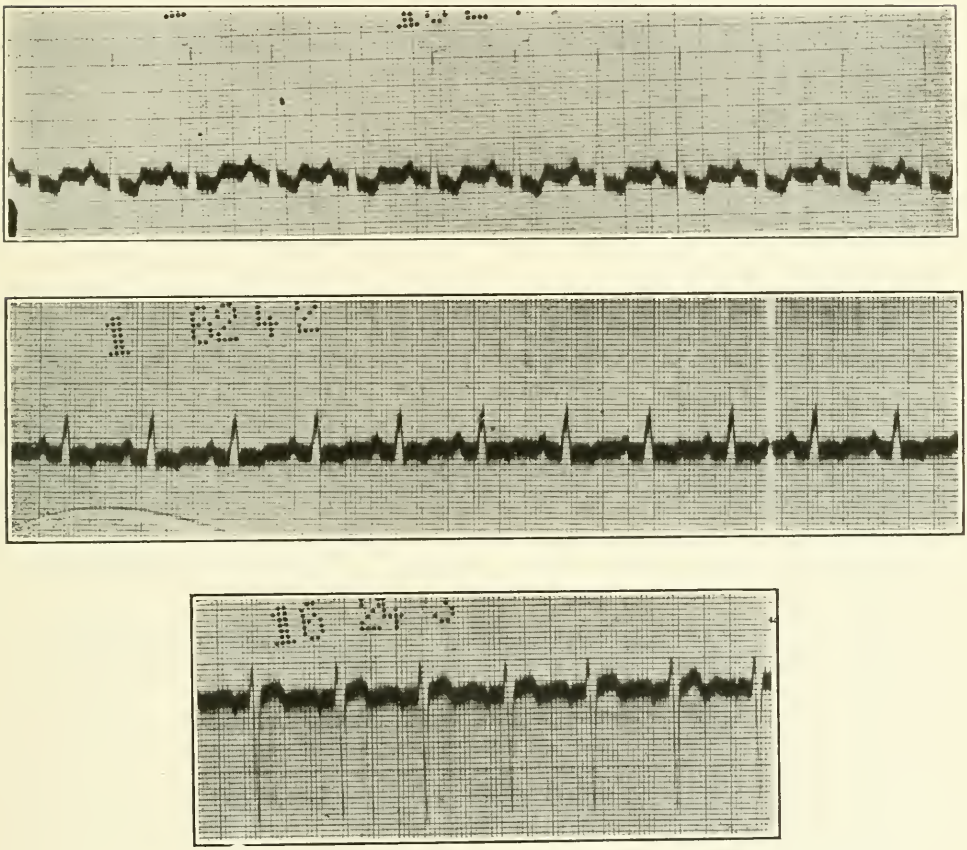


Fig. 3.—The electrocardiogram shows marked left ventricular predominance, notching of R in lead 2, inversion of T in all three leads.

enlarged extending to the iliac crest. No palpable epitrochlears present. The teeth, although suggestive, were not typically Hutchinson's teeth. There were no other stigmata of syphilis.

This case is presented because of the great rarity of aneurysm of the thoracic aorta in children. Theodore Le Boutellier made the most

exhaustive study in 1903. He collected and reported fourteen cases in children under 16 years. In all, we have been able to collect about twenty cases in children. The etiology given in the majority of cases is very indefinite. In one, abscess formation in the thorax and secondary involvement of the aorta is given. Trauma and congenital valve defects are recorded in others. Rheumatism and bacterial infections are also mentioned. In only one case, that published by Wilson in 1913, is there recorded a definite family history of syphilis, but no Wassermann report is given. This case is the first in a child of an unquestionably proven syphilitic aneurysm as far as we have been able to learn.

The patient has had eight inunctions of 2.0 gm. unguentum hydrargyri and up to the present time no apparent improvement has been noted. Salvarsan has not been given on account of a possible danger in this type of case.

I wish to express my appreciation to Dr. B. S. Oppenheimer, in charge of the cardiograph work at Mt. Sinai Hospital, for the tracings.

CASE 1.—Smith, R. T.: Lancet, 1834, p. 626. Aneurism of ascending aorta in 16 year old boy; typical signs—rupture and instant death, cause unknown.

CASE 2.—Miguel: Bull. gen. de thérap. **9**:393. Aneurysm in 14 year old boy; ascending aorta; heart normal in size. Boy indisposed for eight days only before onset of acute symptoms.

CASE 3.—Moutard-Martin: Bull. de la Soc. anat., 1875, p. 775. Patient 2 years old. Ascending aorta; heart hypertrophied. Atheromatous patches in aorta.

CASE 4.—Murdock, B., and Welsh, D. A.: Edinburgh Hospital Report **6**: 84, 1900. Ascending aorta, boy aged 12 years. Aortic and mitral valves diseased, vessels normal. Congenital heart disease when 3 years old. Death due to ulcerative endocarditis.

CASE 5.—Berry, S. H.: Brit. Med. J. **2**:1715, 1898. Boy aged 15 years. Ascending aorta. No history of rheumatism or syphilis; no edema; dropped dead while playing cricket.

CASE 6.—Bacelli, G.: Semaine méd. **18**:137, 1898. Boy aged 16 years. Family history negative. Acute articular rheumatism at 15 years. No previous illness; typical.

CASE 7.—Le Boutillier, Theodore: Am. J. M. Sc. **125**: 1903. Girl of 9 years. Brassy cough; slight substernal pain; no edema; ascites, dyspnea, palpitation, headache and vertigo; typical signs; cause unknown.

CASE 8.—Smith, F. J.: Tr. Path. Soc. London **28**:53, 1897. Male, aged 9 years. Descending aortic arch.

CASE 9.—Hutchinson: Tr. Path. Soc. London **5**:104. Girl 4 years old. Arch of aorta. Mr. Hutchinson believes aneurysm originated in an abscess and had ulcerated into the vessel.

CASE 10.—Roger: Bull. et mém. Soc. méd. d. hôp. de Paris, 1865. Child of 10 years. Suffocative attacks began two years previous; typical signs; tumor above and to the left of sternum, arch of aorta.

CASE 11.—Sanne: Rev. Mens. de Malod. de l'Enfance, February, 1887. Boy of 13 years. Aneurysm of arch; dyspnea, aortic valves with vegetations.

CASE 12.—MacKeen, R. A. H.: Med. News, 1892. Girl 4½ years old, arch of aorta pulsating; swelling in first intercostal space just to left of upper extremity of sternum; expansile pulsation.

CASE 13.—Moore: Tr. Path. Soc. London, 1882-1883. Girl of 5 years. Died of tuberculous meningitis. Heart growths in tricuspid, mitral and aortic valves. Shallow aneurysmal pouch in posterior wall of aorta due to acute inflammation of inner coat of vessel.

CASE 14.—Rie, Oscar: Mitth. der Wien.-Med. Dot. Coll. **15**:134, 1889. Girl of 5 years; typical signs of aneurysm of aorta; dyspnea on exertion; unequal pulse; etiology unknown.

CASE 15.—Pendin, A.: St. Petersb. med. Wchnschr., 1890, p. 195. Girl of 12 years; typical symptoms and signs. Etiology due to a fall six years previously.

CASE 16.—Modsen, S. I.: Med. Rev. Bergen **28**:530, 1916. Child of 17 months. Rupture of an aneurysm of aorta.

CASE 17.—Willson: Proc. Path. Soc. Philadelphia **16**:63, 1913. Child of 4 years. Specimen obtained from a child who died suddenly following several months of grave anemia and glandular enlargement of syphilitic origin. No Wassermann report.

CASE 18.—Roy, P.: Bull. et mém. Soc. anat. de Paris **28**:198, 1908. Boy 15 years. Typical signs—ascending posterior; cause unknown—infection of some kind.

CASE 19.—Horder, T. J.: St. Barth. Hosp. Rep., London **43**:57, 1908. Boy of 12 years. Typical sign at necropsy; ascending posterior; cause not known; rupture.

CASE 20—Lance, Jordan: 1903. Boy of 6½ years. Ascending arch; history of acute rheumatic fever; rupture of sacculated aneurysm of ascending arch; possibly a septic aortitis.

64 West Eighty-Fifth Street.

REPORT OF A CASE OF TYPHOID FEVER IN INFANT OF 8 MONTHS TRANSMITTED THROUGH THE BREAST MILK OF THE MOTHER

HENRY HEIMAN, M.D.
NEW YORK

March 19, 1919, there was admitted to the adult medical service at Mount Sinai Hospital a nursing mother suffering from typhoid fever. Two days later the patient's baby was brought to the pediatric service, afflicted with the same malady. A bacteriologic study of the mother's breast milk disclosed the presence of typhoid bacilli.

HISTORY OF THE MOTHER'S CASE

The patient was a married woman of 29 years, whose family history and past history were quite negative except for an attack of influenza six weeks previously. Two weeks before admission onset with pain in lower back region, headache, fever and chills. No epistaxis. Diarrhea for several days. On examination patient was found acutely ill, restless and actively delirious. Lower pole of spleen felt 3 inches below costal margin. Definite rose spots present. Pulse was dicrotic. Temperature while in the ward showed typical curve, gradually falling in the fifth week of the disease from 105 degrees to normal. The blood showed a leukopenia, the white count being 4,200 with 68 per cent. polynuclears and 32 per cent. lymphocytes. The blood culture showed the presence of typhoid bacilli and the Widal reaction was positive. A culture of the breast milk revealed the presence of actively motile bacilli corresponding in every respect to the typhoid organism.

HISTORY OF THE INFANT'S CASE

The patient was an infant of 8 months whose birth history was perfectly normal. It was breast fed until two weeks before admission. (Weaned because of mother's illness.) There was no previous illness or physical defect. Four weeks before the admission to the hospital onset with rise of temperature to 101 F., but infant seemed as well as usual. In ten days it had apparently recovered entirely. Then there followed a sudden rise of temperature to 105 F. Both ear drums were incised, but the fever continued. No vomiting or diarrhea. No convulsions. On examination baby appeared acutely ill with respirations rapid, and cheeks flushed. There were no rose spots. The spleen was enlarged, the lower pole being felt 1 inch below costal margin. The rest of the physical examination was negative. The blood count was 8,000 white cells, with 74 per cent. polymorphonuclears and 26 per cent. lymphocytes. The blood culture showed the presence of typhoid bacilli and the Widal reaction was positive.

We have found the experience worthy of report because of its very unusual occurrence. We have been unable to find any similar record in

the literature. In one case, however, reported by Lawrence[1] typhoid bacilli were found in the breast milk in a nursing mother of 23 ill with the disease. But in this instance the infant escaped infection.

I wish to thank Dr. N. E. Brill for permitting me to use the material from the adult service at Mount Sinai Hospital.

DISCUSSION

DR. SMITH: Dr. Charles H. Lawrence of Boston reported in 1908 a case in which he found typhoid bacilli in the breast milk. The patient was admitted to the Massachusetts General Hospital with typhoid fever and the bacilli were isolated from the breast milk at that time.

DR. HEIMAN: I just want to apologize for presenting this as the first case in which typhoid bacilli were found in human breast milk. I shall look it up and shall be very glad to add that to my report.

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1. Lawrence, C. H.: Boston M. & S. J. **151**:153 (July 29) 1909.

SOME DETAILS IN THE MANAGEMENT OF THE PREMATURE INFANT (Abstracted)

ISAAC A. ABT, M.D.
CHICAGO

The case report which follows is of a premature baby who was born about ten weeks before term. It was a twin pregnancy; the boy was born dead, and the girl survived. Her weight was about 3 pounds 7½ ounces. The case is made the subject of a special report on account of the minute records which were kept, the careful observations which were made and the exact and painstaking attention to detail.

The baby was fed breast milk. For the first few days she was fed every two hours; from the sixteenth day every three hours; and subsequently she was put to the breast and fed every four hours. She was receiving about one fifth of her body weight in food and was gaining well.

Particular interest attaches to the paintaking effort directed to maintaining temperature conditions. The baby was placed in an incubator, and three thermometers were placed in the incubator, one at the head of the infant, one at the foot and one in the bassinet. The temperatures of the three thermometers were read at the same time and varied considerably. The temperature read from the thermometer within the bassinet constituted the important reading, and was maintained at 77 or 78 F. for the first two weeks. The temperature of the room was carefully controlled. More than half a dozen thermometers were distributed throughout the room, and readings were made every fifteen minutes. The room temperature was kept at about 74 or 75 F. Eventually, however, it was necessary to reduce the room temperature to 71 or 72, and when it was desired to reduce the crib temperature to 74, the room temperature was maintained at 70.

It was found that the room was most comfortable when the humidity reached 49 or 50 per cent. It was found that this degree of moisture could be maintained best by moistening Turkish towels and hanging them on screens. This seemed to be the most effective method because the towels maintained the moisture for a longer time.

The baby gained steadily in weight, and although it developed a slight craniotabes and a moderate hydrocephalus, it went steadily forward, and at six months of age weighed as much as a baby which was normal at birth. The hydrocephalus did not increase, and the craniotabes disappeared. (Lantern demonstration.)

CONGENITAL DISSEMINATED ATELECTASIS WITH ROENTGEN-RAY FINDINGS: RECOVERY

JOHN RUHRAH, M.D.
BALTIMORE

My only reason for making this brief report is that in the experience of at least three of the roentgen ray establishments in Baltimore not a single instance of this condition had been discovered. The patient was a girl born July 27, 1917, weight 6 pounds and 7 ounces at birth; this increased up to 8 pounds, at which it remained. The baby was nursed two months and then weaned and placed on a large number of different milk formulas and preparations. When first seen, Nov. 15, 1917, she had already passed through the hands of four doctors. The case had been regarded as a feeding case, but on examination the patient showed a certain amount of cyanosis that became very marked on crying. There was marked cyanosis and clubbing of the tips of the fingers and toes, a curious duskiness of the skin of the abdomen with enlarged superficial veins running up the chest. The most striking thing, however, was the rapid respiration, which rarely was under 50 a minute and many times 75 or 80 or more. The heart was normal as far as could be determined, and nothing could be made out in the lung. The child, with proper care and attention to feeding, gradually gained, and in June, 1918, weighed about 11½ pounds and was breathing somewhat more slowly.

At present the child weighs 25 pounds and is apparently normal. The roentgenogram, taken by Dr. Frederick H. Baetjer, reveals the disseminated patches of unexpanded lung. The areas vary in size from a pin point to a grain of wheat, scattered through both lungs, but are more numerous in the left and are most sparingly distributed in the outer portions of the lung. A second plate taken recently reveals a normal chest.

DISCUSSION

Dr. Heiman: What were the physical signs that corresponded to the roentgenogram?

Dr. Ruhräh: There were no physical signs, just the clubbing of the fingers. The heart was normal. We had about a dozen pictures taken, but could make nothing out except the roentgen-ray findings. The light box shows disseminated areas in both lungs that cleared up in about a year's time.

A CASE OF CALCULUS

PERCIVAL J. EATON, A.M., M.D.
PITTSBURGH

Harry S., 6 years old, was first seen, Nov. 28, 1918, giving then a history of incontinence by day as well as night. He had had a mild attack of influenza, about two months earlier, which lasted two or three weeks and which seemed to have pretty well cleared up. At the time of his visit to our office he did not complain of pain on urination. Before coming to the office he had been circumcised, with no relief to the incontinence. His previous medical history disclosed measles, mumps and chickenpox, none of which seemed to have any bearing on his present condition. Dry diet the latter part of each day, and the elevation of the foot of the bed at night seemed to have no effect, and he soon began to complain of much pain on urination. The pain was severe, paroxysmal in character, and the child would grasp his genitals and "hold himself," trying to mitigate the spasm of pain. The urine showed nothing pathologic. As the boy lived in a neighboring town, and as most of our knowledge of the progress of the case came from telephone reports, we had him admitted to St. Margaret's Hospital, Jan. 7, 1919.

The urine on admission was straw colored, turbid, slightly acid, containing a faint trace of albumin, but no glucose. A few leukocytes, a few epithelial cells and many bacteria made up the sediment. The specific gravity was from 1.010 to 1.020. The child's periods of pain were very irregular, and manipulation of his bladder area would often excite a paroxysm. The pain was so severe at times as to necessitate the use of codein. After some delay we obtained two roentgenograms, which revealed a good-sized oval stone in the bladder. Its presence was confirmed by the sound.

The patient was transferred to the surgical side and operated on by Dr. J. D. Singley. A suprapubic incision was made into the prevesical region, the peritoneum was pushed away, and an incision made into the bladder large enough to allow of the delivery of the stone, which weighed 18.25 gm., and which is herewith presented for examination. The boy made an uneventful recovery.

The points of interest are:

1. Incontinence of the usual type, probably resulting from the influenza.

2. Absence of pain at first.

3. Paroxysmal pain becoming more and more severe.

4. The size of the stone, with no apparent reason for its occurrence, i. e., no history of the child's having lived where the water was very hard; no history, indeed, along any line.

5. The probable long time of its existence in the bladder without symptoms.

A CASE OF SCORBUTIC HEMATURIA

PERCIVAL J. EATON, AM., M.D.
PITTSBURGH

March 19, 1919, I was called by Dr. Graham of Sharpsburg, Pa., to see a case of suspected stone in the bladder (Baby Fair). The child had had a hematuria for two weeks, and had been seen by another physician, as Dr. Graham was still in service. The history was not at all complete, but was that of a rather poorly nourished child, fed for a long time on patent foods, and then on cooked food to some extent, but never fed on fresh, raw food. It had no scorbutic symptoms so far as its gums were concerned, though they were unduly red. The child was not particularly sore on being handled. His condition seemed to me to point more toward scurvy than to calculus, especially as he had no pain, and more especially in that the urine showed nothing abnormal except bright blood. I advised fresh, raw milk, beef juice and orange juice; and against the use of the sound until at least antiscorbutic treatment had been given a fair opportunity. Dr. Graham later phoned me that the hematuria disappeared rapidly under the advised treatment and that the child made a rapid and complete recovery.

As this was the second case of scorbutic hematuria which I had seen, I was much interested to have my diagnosis confirmed. I was too busy to look up the literature in the short time at my disposal, but such cases are rare, I believe.

A CASE OF ANOMALY OF THE DIAPHRAGM

WITH HERNIATION INTO THE THORAX OF CERTAIN VISCERA RESULTING IN A GASTRIC AND INTESTINAL OBSTRUCTION

L. R. DeBUYS, B.S., M.D.
Professor of Pediatrics, School of Medicine, Tulane University
NEW ORLEANS

REPORT OF CASE

Dorothy C. was admitted into my service at the Touro Infirmary May 2, 1919. She had been ill two days prior to admission and was sent to the institution by a physician with the diagnosis of "locked bowels."

Past History.—She was four months and five days old, and was the only child of apparently healthy parents. There was no history of miscarriage and the delivery of the patient was normal, except that the cord was wound around the neck. No instruments had been used. She had had no "blue" spells, and had been perfectly well and healthy up to the age of three months when she had a peculiar attack of colic with twisting and straining. This "spell" was promptly relieved by the administration of milk of magnesia and an enema, after which she "got all right." Another similar attack occurred between the first one and the one for which she was admitted. It was a little more pronounced than the first, but she promptly recovered from it also, the same treatment being employed.

Present Illness.—Two days previous to admission her parents had gone on a trip to the lake shore, and while there the patient began making a grunting noise. On the way home a "weak spell" occurred, and on reaching home the baby wanted to vomit, but could not. From this time on she could not nurse. What was taken would come up immediately as it went down. Her bowels, however, moved this day. The next day the grunting continued, becoming worse as time passed. Attempts were made as on the two previous occasions to overcome the condition, but it was impossible to give anything by mouth as she would "not swallow it," and enemas brought back nothing. She became progressively worse and was in a desperate condition when I saw her for the first time.

Physical Examination.—Dorothy was an apparently well developed and well nourished baby, acutely ill, with a rapid respiration, 78 per minute, and a pulse of from 150 to 160 per minute. Her temperature was 103 F. and rapidly rose to 104 F. The respirations were suggestive of a pneumonia, being jerky, and with a respiratory grunt, accompanied with movements of the alae nasi. On inspection, the chest showed less movement on the left side both anteriorly and posteriorly, and it was apparently not as full as on the right side. Palpation revealed less fremitus on the left side as compared with the right side. On percussion the resiliency of the right thorax seemed normal, but it was absent on the left side, and the notes elicited were different. On the right side the note was apparently normal or probably a little hyperresonant, while on the left side it reminded one of a note with an element of tympany. On auscultation, the voice and respiratory sounds on the right side were distinctly puerile,

while on the left side they were absent. There were no râles. The heart was displaced to the right, the apex beat was in the median line on its normal level.

The abdomen was flat and soft, and presented a tumor in the epigastric and left hypochondriac region. It was lying transversely, was about the roundness of a small banana, and seemed to be from three to four inches long. It was distinct from the lower border of the ribs. It was immovable. The liver was normal; the spleen could not be differentiated from the tumor. Otherwise the abdominal examination, as well as the rest of the physical examination, was negative.

A digital examination was made which showed no tumor in the pelvis nor in either of the regions of the descending or ascending colon, but there was a tumor conforming to the one already described, slightly boggy, and in places giving the sensation of fluctuation.

Blood Examination.—The blood examination showed a total leukocyte count of 29,500 with an 88 per cent. polymorphonuclear-neutrophilic cell count.

A bismuth enema was given with the baby in an inclined plane of about 30 degrees, head down. Only a small quantity of the enema could be given, as the baby would expel it promptly and at regular intervals, so that the time was judged and just before the enema was expelled a roentgenogram was made (Fig. 1). This clearly showed an obstruction. The roentgenogram suggested an intussusception. Remembering, however, the physical findings of the chest with the displacement of the heart to the right, the thorax in the picture was read. This showed a decided shadow on the entire left side. As the shadow was on the side where the physical findings were decidedly against even the suggestion of a pneumonia, and as the duration of the illness was hardly sufficient to have produced an empyema, and as an empyema sufficient to produce a displacement of the heart would at least not have given to the thorax an appearance of being not as full as the opposite side, the surgeon, Dr. Russel E. Stone, who was called in for operation for the obstruction, was requested to look for the possibility of a diaphragmatic hernia. This was shown to exist at the operation.

Operation.—Assisted by Drs. I. M. Gage and J. D. Rives, and with the patient under ether anesthesia administered by Dr. A. B. Pitkin, Dr. Stone performed the operation which he describes with the findings as follows:

"An incision was made in the midline extending from the umbilicus to about an inch above the symphysis pubis. The peritoneum was opened showing the usual amount of clear, straw-colored fluid in the abdomen. The intestines were found empty and contracted, and of normal color; the peritoneum was glistening and not congested; no omentum was visible. Palpation under the upper angle of the incision revealed a tense fluid tumor under the left costal arch, about the size of an ordinary goose egg; inspection showed this tumor to be of the color of gangrenous bowel with blood vessels standing clearly on the surface; some hemorrhagic areas were also visible. On palpating the tumor there was a sudden gush of a blood tinged watery fluid through the nostrils and mouth; at this point there was a cessation of breathing, and all attempts at resuscitation were futile. The incision was then prolonged upward to the left of the umbilicus, almost to the xiphoid; the great omentum and transverse colon were still not visible. The spleen was now seen lying on top of the tumor, with its long axis transverse to the abdomen and its convex surface lying against the diaphragm, and the lower edge just under the left costal arch. It was intimately adherent to the tumor on its anterior aspect.

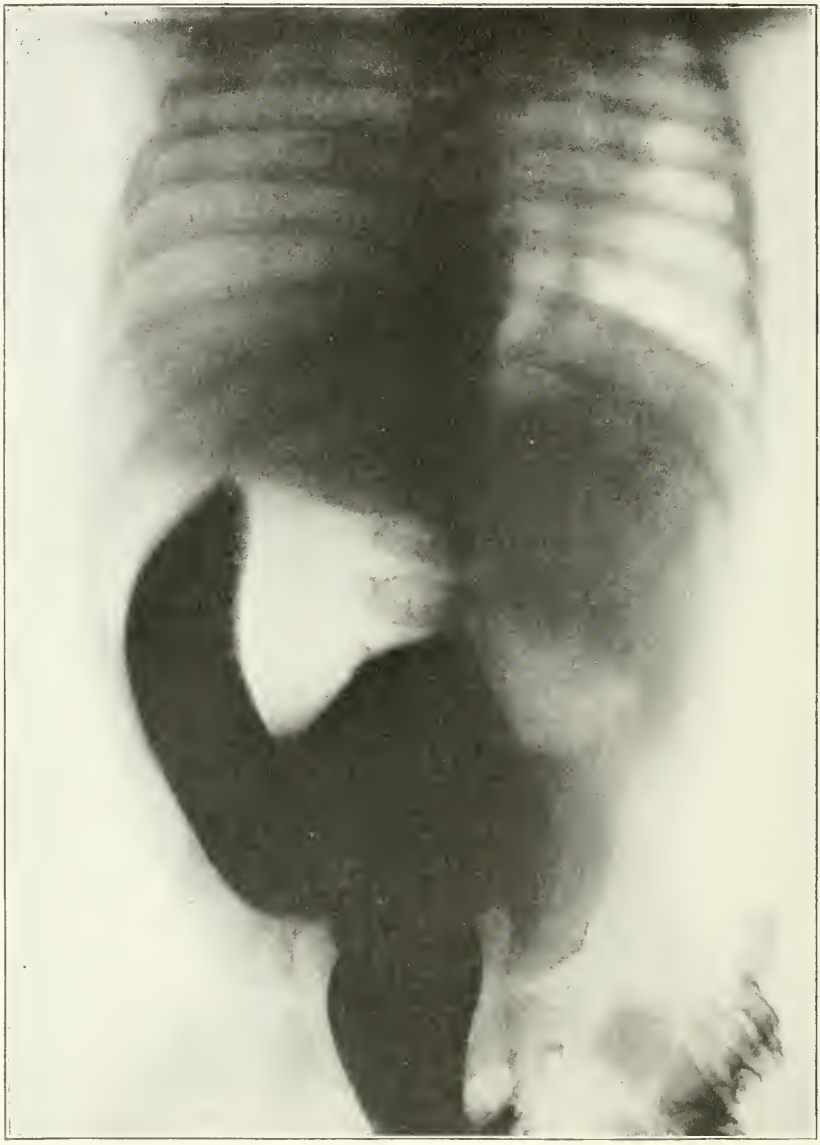


Fig. 1.—The shadow in the left side of the chest is quite apparent, and inasmuch as the heart was displaced to the right, a diaphragmatic hernia was suspected.

"A complete exploration of the upper abdomen was now made; the liver and gallbladder were found to be normal, but the tumor was attached to the hilum of the liver; the transverse colon was still not in sight. Digital examination revealed a hiatus in the diaphragm under the tumor which was distinctly felt to be continuous with another fluid mass in the thorax. The incision was now prolonged upward to the left of the sternum, the costal cartillages were divided, and the left chest opened revealing the fundus and the

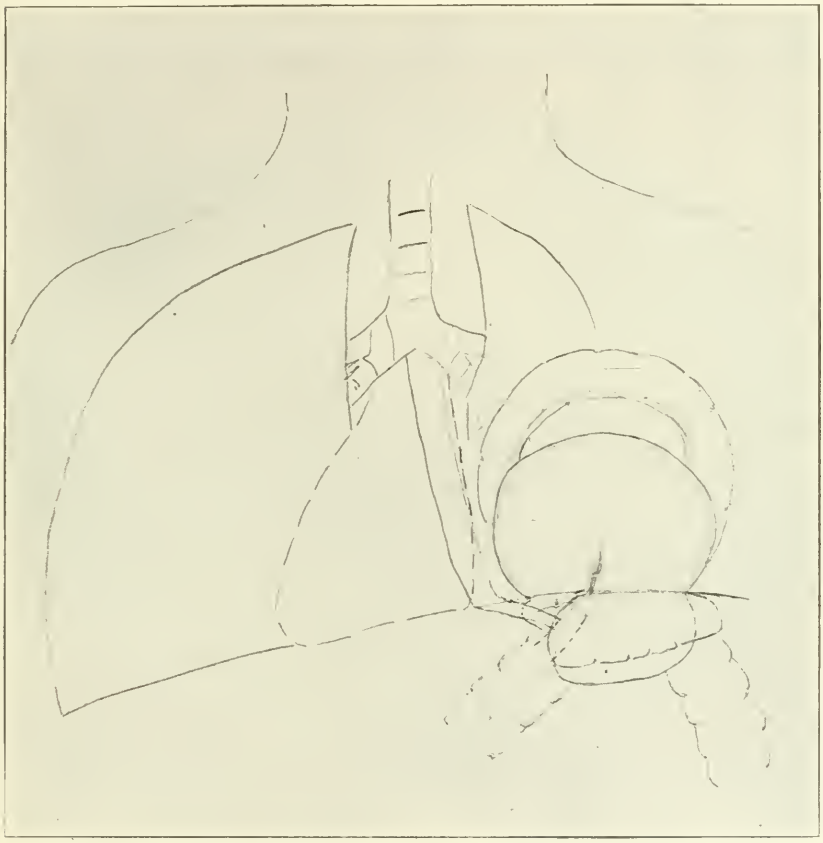


Fig. 2.—Schematic relations of viscera in thorax and in abdomen.

greater part of the body of the stomach, the transverse colon and great omentum in the thoracic cavity. No pleural sac was found over these viscera, they were of normal color; the stomach was distended and the colon empty. (Fig. 2.)

"The diaphragm was divided down to the hiatus. The tumor consisted of the pyloric portion of the stomach twisted on its axis at the hiatus behind the

diaphragm, so that the greater curvature with the attachment of the gastrosplenic omentum lay in contact with the inferior surface of the diaphragm, under the left costal arch. Thus the pyloric portion of the stomach was strangulated and the pylorus closed by torsion, while the portion of the stomach in the chest cavity was not strangulated. The transverse colon and omentum had slipped up behind the stomach.

"The left lung was collapsed and showed no evidence of pneumonia. The heart was displaced to the right so that the apex lay on the spinal column at about the fourth chondrosternal junction. The wound was closed with two layers of continuous catgut sutures."

Diagnosis.—Diaphragmatic hernia containing the stomach, transverse colon and great omentum.

Being desirous of more information with regard to the diaphragm and left thorax and the displacement of the heart, after much persuasion a partial necropsy was granted by the parents under the agreement that the body, the abdomen and thorax only, were to be inspected without removal of any tissue, and that the necropsy was to be done through the wound resulting from the operation. The necropsy was performed by Dr. C. W. Duval, whose report is as follows:

NECROPSY REPORT

The body was that of a baby well developed and well nourished, about four months of age. The interior of the abdomen and thorax were exposed by opening the wound resulting from the operation, which extended from slightly below the umbilicus upward to the right of the xiphoid process; the intercostal cartilages of the lower ribs had been incised. A good view of the abdomen and thorax was obtained. On inspection, the lower end of the esophagus and the first portion of the duodenum were found to be ligated and the stomach and spleen missing; the latter had been removed at the time of the operation after the death of the child. The left suprarenal was found to be enlarged and hemorrhagic, and was removed for microscopic study. The small and large intestines were normal.

The diaphragm was next examined very carefully; the normal openings (vena caval, esophageal and aortic) were intact and presented no defects; its attachments were normal, except in its posterior left half. From a point 6.5 cm. from the vertebral column, for a distance of 5 cm. at the level of the sixth rib (at which point it became attached again), there was no attachment; there was a hard cord bridging over this area in a dome-like manner. This section of the diaphragm was removed.

The left lung was found to be atelectatic, except for a small portion of its periphery. The heart was displaced to the right side; the apex was beneath the sternum. The right lung was found to be perfectly normal and had been functioning properly.

The stomach appeared bent on itself at a point from above downward beginning at the esophageal end anteriorly. There was another twist that appeared to be anterior to the pyloric orifice. The twisting in these two portions had cut off certain blood supplies which resulted in gangrenous necrosis

of the stomach wall. There were three different areas of gangrenous necrosis. One was on the anterior surface of the stomach beginning along the lesser curvature at the pylorus and extending to the esophageal orifice; this area extended down over the anterior surface of the stomach in a fanlike manner for a distance of 5 cm. The second area of gangrenous necrosis appeared posteriorly and was about the size of a 50-cent piece; its situation was near the lesser curvature. The third gangrenous area, the longest of the three, included almost all of the fundus for the anterior surface of the stomach.

The transverse mesocolon and the gastrosplenic omentum also showed definite areas of gangrene. The stomach contained much coagulated and liquid blood.

Fig. 3.—Diaphragm seen from below upward. Error of attachment on left side.

The spleen, aside from marked congestion, appeared normal.

The section of the diaphragm removed for study showed the band to consist of a crude cordlike mass, thicker in the center than at its attachment ends. The cross section measurement at the center was 4 mm. and at the ends 2 mm. This cordlike bridge was perfectly smooth and glistening in its entire extent.

Microscopically, the cross section of the cord showed a central mass made up of aggregated bundles of muscle fibers for the most part running in one plane along the length of the cord. There were, however, those bundles that

were in other planes running more or less oblique to the long diameter of the cord. This central core of muscle bundles was enveloped for 90 degrees of the arc by a normal zone of connective tissue; over this zone of connective tissue there was attached the pleural and peritoneal serous membranes.

Throughout the pancreas there was extensive necrosis both of the acini and the Langerhans islands, except in a few small areas at the tail, which were in a normal state of preservation. The necrosis of the pancreas had undoubtedly resulted from a twisting of the organ in such a way as to hurt its blood supply. The absence of fat necrosis in the abdominal tissues at necropsy further indicated that the destruction of the pancreas was quite recent.

The left suprarenal showed also that its blood supply had been interfered with because of the extensive hemorrhage that appeared in the medulla. The cortical tissue appeared not affected. The right suprarenal was negative.

DISCUSSION

This case showed many points of interest. The two previous attacks were probably caused by a slight involvement of the intestine through the abnormal opening, but the fatal attack apparently began with an involvement of the stomach and later because of the straining the colon became complicated. In substantiation of this fact may be offered the gangrene of the stomach and the normal tissues of the intestine found at operation. Again the bowels discontinued moving the day following the onset of the attack showing the colon to be free later than the stomach; also there was no distention of the intestines either small or large, due to the fact of their having become relatively emptied after entrance of food from above and exit of the food from below before obstruction of the colon.

The obstruction of the stomach is of extreme interest both because of the pressure with occlusion at the cardia and the twisting of the pylorus with its obstructon at the point of entry into the thorax. The fatal termination was probably due to shock. The blood-tinged watery fluid which came through the nostrils and mouth was part of the contents of the stomach expelled when the pressure at the cardia was relieved. While the baby died of shock at the time of operation it is hardly probable that the outcome of the case could have been different because of the physical condition of the patient, the duration of the obstruction, and the gangrenous condition of the stomach.

The absence of tenesmus with bloody stools in an obstruction involving the descending colon should require the exclusion of the diagnosis of intussusception. The roentgenogram without any evidence of air in the thorax, especially with an intestinal herniation into

it and without any shadow due to bismuth, is of decided interest (Fig. 1).

This hernia belongs to the false variety of the congenital type of diaphragmatic hernia being differentiated from the true variety by not having a sac. In this instance there existed two conditions not mentioned in the various classifications of diaphragmatic hernias, namely, strangulation and palpable abdominal tumor.

The defect of the diaphragm (Fig. 3) in this case was a developmental error which occurred at the point at which in the developmental progress of the membranum transversum the attachment is completed last, namely, on the left side and posterior aspect.

ABSTRACT OF REPORT OF A CASE OF TRICHINIASIS

HOWARD CHILDS CARPENTER, M.D.
PHILADELPHIA

History.—J. L., Italian boy of 13, had had good health until taken suddenly ill three months before coming under my observation, with diarrhea, vomiting and pain in the epigastrium. After eight days of irregular fever, the pains became general, with swelling of the muscles, which were painful to pressure or on motion. These symptoms were accompanied by itching of the skin. The face became swollen and within ten days the boy was so ill that he was unable to raise his head from the pillow or to move his legs. Other symptoms observed were marked irritability, constant picking of the nose and a persistent cough. The prostration had been marked and the child had been confined to bed, according to the history, for six weeks. He had been treated for various conditions, such as muscular rheumatism and growing pains.

The chief complaint after three months of illness is pain and soreness in his muscles. The physical examination showed an anemic child with malnutrition, 10 pounds under weight. The voluntary muscles on first inspection looked well developed, but on closer examination these muscles were found to be swollen and tender. They were firmer than normal, but this firmness was not uniform; that is, the muscles had a lumpy feel. These infiltrations in the muscles varied in size, and were felt in the masseters, the biceps, triceps, and the gastrocnemii. The calf muscles, hamstring and lumbar muscles exhibited a distinct state of spasm. This spasmodic condition was most marked in the calf muscles, causing the boy to walk on his toes when his shoes were removed. The child complained of a pulling pain in the sacrum, and when seated on the floor with the legs extended, if the knees were pressed down to the floor he would fall backward. His muscular strength was decidedly subnormal. There was slight edema over the tibia.

The eyes were prominent, pupils dilated, and the pupillary reactions were sluggish. Stellwag's and von Graef's signs were present.

The superficial glands in the axilla, groin and neck were just palpable. The examination of the heart revealed the right border to be in the right parasternal line, and the left border in the left midclavicular line. The muscular quality of the first sign was below normal and a soft systolic murmur was present, heard best at the apex. His pulse was of fair volume, regular, and the rate at the time of the examination was 94. From the history obtained, the boy's pulse had been running from 110 to 150 during the early part of his illness. The blood pressure was systolic 110, diastolic 92.

The examination of the lungs was negative. The abdomen was somewhat prominent; this prominence was due largely to a lumbar lordosis. The liver extended 2 cm. below the edge of the ribs in the right midclavicular line. The spleen was easily palpated 0.5 cm. below the edge of the ribs. Only a very slight knee jerk could be obtained. There was no Achilles' tendon reflex. Kernig's sign was present. The joints showed no enlargement, pain or tenderness. When questioned as to where he had the most pain, he complained of the neck muscles.

The blood examination showed a hemoglobin of 70 per cent., a red cell count of 3,002,000, and a leukocyte count of 16,000. The differential count was neutrophils 43 per cent., lymphocytes 36 per cent., large mononuclear leukocytes 5 per cent., transitionals 1 per cent., and eosinophils 15 per cent.

The patient came too late under our observation to make it worth while to examine the blood for the parasites, as they have not been found in the blood after the twenty-fifth day of the infestation.

Examination of the feces failed to reveal, as would be expected, any evidence of trichina, and careful repeated searches for ova of other intestinal parasites were negative. An attempt was made to determine an intestinal eosinophilia, but without success.

Several urine examinations gave uniform results: a specific gravity of from 1.010 to 1.014, acid reactions, no sugar or acetone, no excess of indican, and faint traces of albumin. The microscopical examinations were negative except for slight excess of pus cells and bladder epithelium.

The history of the infection was obtained with difficulty, because of the ignorance of the family. It was ascertained that three days before the boy was taken sick, he had eaten at the home of his uncle some raw homemade sausage. At this meal, besides the patient and his brother, there were an uncle and an aunt and their three children; a boy of 13 years, and two girls, aged 11 and 7 years. A day or two later the aunt and the two girls had been taken acutely ill with vomiting and diarrhea, and were sent to a hospital, with a diagnosis of ptomain poisoning. They remained in the hospital for only one week, but because their condition was unimproved they were taken home. The aunt died in a week after her return and the girls remained sick in bed for about eight weeks. From the description which I obtained of their illness, the symptoms were so like those of my patient that I had the two girls' blood examined, and also the blood of the uncle and his son, although at first he denied that either of them had been sick. The blood of the 11 year old girl contained 20 per cent. eosinophils; that of the 7 year old girl, 26 per cent. eosinophils; that of the 13 year old son, 9 per cent. eosinophils; and that of the uncle 10 per cent. eosinophils.

After repeated questioning the uncle admitted that both he and his son had had, at the time that the other members of his family were sick, an attack of diarrhea, followed later by some pains in the arm and leg muscles, and that these muscles had been slightly tender. The small brother of my patient, who also ate of the sausage, had an eosinophilia of 14 per cent., although his mother absolutely denied that he had been sick.

By the time I first saw my patient, after three months of illness, the infestion must have been in the third stage, that of regression. The invasion of the muscles was finished and the embryos encapsulated, but there had not been sufficient time for calcification of the capsules. The absence of the calcification was confirmed by a series of roentgenograms of the muscles, which were entirely negative. (In order to hasten calcification treatment by the roentgen rays would seem to be indicated at this stage.)

Because of the severity of the attack in this boy, in spite of the early protective diarrhea, the pork which he ate must have been heavily infested. From the history of my patient and of the other infested individuals in this family epidemic, the statement that the disease is mild in children has not been borne out. Three of the four children infested had been extremely ill, judging not only from their history, but from their physical condition, now three months after infection.

I believe that not a few cases of so-called chronic muscular rheumatism in children are really chronic trichiniasis. Trichiniasis should not be overlooked in this delicatessen age, when the mother on her way home from the "movies" all too frequently stops and purchases bologna sausage for the family's supper.

These cases fall in line with the largest percentage of the reported cases, in that the pork which they consumed was in the form of sausage, and that the sausage was home made.

We were able to obtain a piece from the sausage which had been the cause of the infestation, but a painstaking search of over fifty slides failed to reveal any trichinae. We have not had sufficient time yet, but hope to feed some of this sausage to rats, and in this way further confirm the diagnosis. If we are successful in infesting one or more rats, we will make a thorough dissection of their muscles in an effort to collect a sufficient number of trichinae to make an antigen, with which to do a complement fixation on the patient, and on the other infected individuals. As far as I have knowledge no complement deviation tests have been tried in trichiniasis, although it has given positive results in *Tinea saginata* infestions.

It is interesting to note that this child three months after the infestation still had a decided increase in his leukocytes (16,000), and still had a marked eosinophilia (15 per cent.); that is, an absolute as well as a relative eosinophilia. This fact is one of such importance in confirming the diagnosis that it is practically pathognomonic when infection with other parasites can be excluded by failure in repeated attempts to find their ova in the feces, and in the absence of any parasitic skin disease.

DISCUSSION

DR. ABT: Did the child have any edema?

DR. CARPENTER: The child still had a slight edema at the time I saw him. His history showed that he must have had considerable edema and pallor before that time.

TWO CASES OF PYELITIS AND ONE OF SUPPRESSION OF URINE FOR FOUR DAYS

ROWLAND G. FREEMAN, M.D.

The first case I wish to report is interesting on account of the peculiar reaction of the child to treatment.

The patient was a girl about 9 months old, who, after doing very well for some time, suddenly developed a fever. This was, as may be seen by the chart, of a remarkably remitting and irregular character, which immediately suggested pyelitis. An examination of the urine showed a moderate number of leukocytes present.

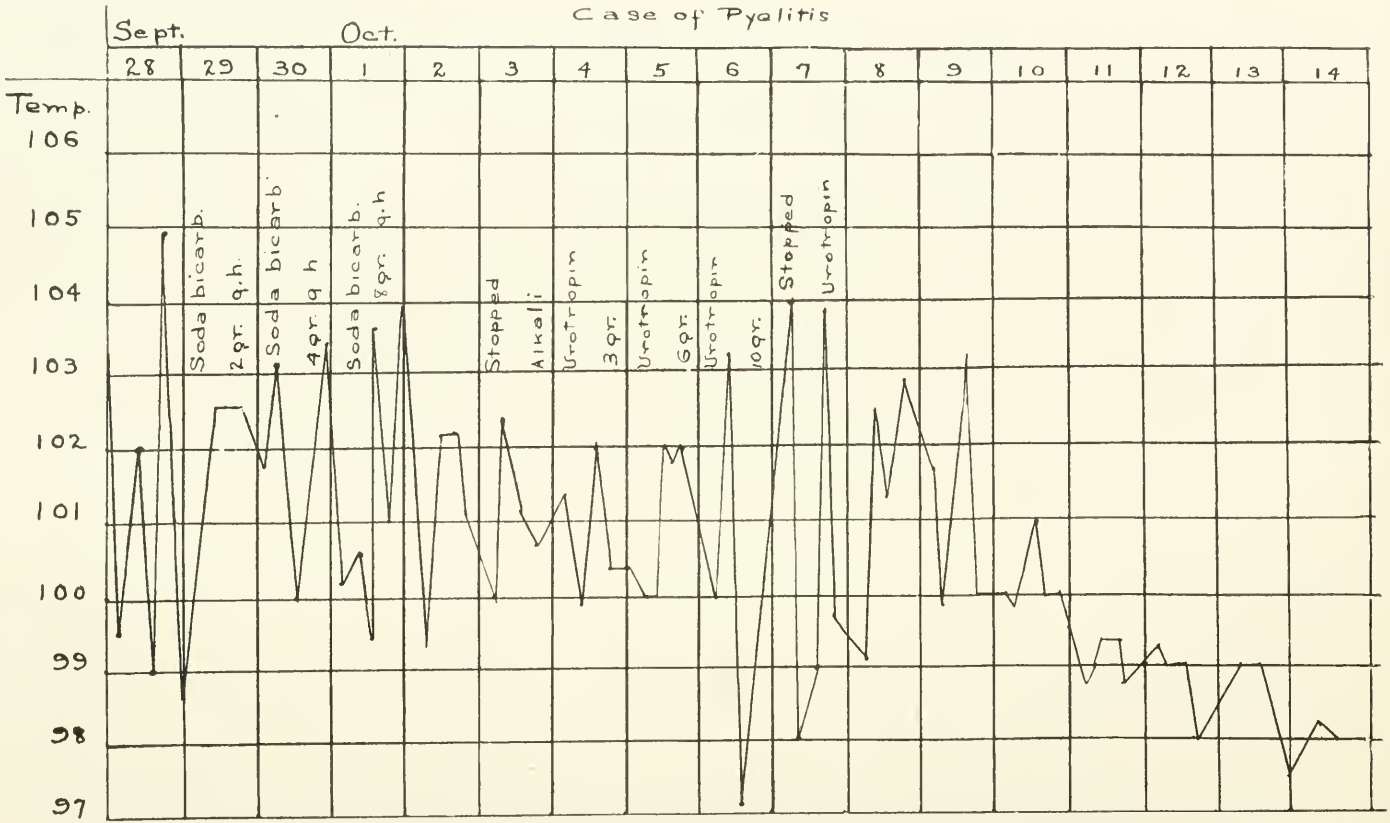
The child was put on alkaline treatment, consisting of the hourly administration of 2 grains of sodium bicarbonate. As the dose was increased the amount of pus in the urine increased markedly, so that while before treatment the urine contained only twenty white blood cells in a D field, the day following the treatment there were 200 and the day after that 200. This occurred with no remission of the temperature and induced me to discontinue the use of the alkali and try hexamethylenamin. During the twenty-four hours after alkaline treatment was stopped the temperature was lower and the number of white cells in the urine was reduced to 50 in a D field. Hexamethylenamin was then administered in daily increasing dose, beginning with 6 grains the first day, when the urine showed only eight white blood cells to a D field; but there occurred on that day an increase of 1.5 degrees in the maximum temperature. With no treatment the child's temperature reached 102; with 6 grains of hexamethylenamin it went up to 103.5, and with 10 grains a day it reached 104.4. The leukocytes in the urine at the same time gradually increased to 150 in a D field, and as the baby seemed much worse, treatment was stopped after four days. On the following day the temperature reached 103 and three days later was normal. The urine, however, continued to show about 200 white blood cells to a D field. The child had no further fever and no more specimens of urine were sent for examination.

Two months later, it having occurred to me that the last specimen still showed leukocytes, we examined the urine again and found the pyelitis still present, although the child had seemed perfectly well in the meantime.

The alkaline treatment was then undertaken, but without clearing the urine. Then hexamethylenamin was given alone and with benzoic acid and dilute hydrochloric acid to acidify the urine, and finally large doses of alkalis were again administered.

After one month of treatment the urine became sterile on a pint of Kalak water daily and 16 grains of sodium bicarbonate every hour day and night, and has remained sterile.

This case is of particular interest because while the child had constitutional symptoms from the pyelitis, each effort to cure the child, whether by the alkaline treatment or by hexamethylenamin, seemed



Temperature chart and daily medication of case of pyelitis apparently made worse by the usual curative treatment.

to make the condition worse both in respect to the fever and the number of leukocytes in the urine, so that the treatment was discontinued. Within three days of the cessation of treatment the temperature became normal and the child apparently recovered, although she was left with a pyelitis without constitutional symptoms. It was finally cured and at this later stage neither large doses of 16 grains of sodium bicarbonate each hour with the addition of Kalak water, nor the large doses of 30 grains of hexamethylenamin daily caused any apparent disturbance of the child's health.

SECOND CASE OF PYELITIS

The second case of pyelitis was in a frail child who weighed only 5 pounds at birth, but who did well until the latter part of the first year when she had a colitis followed by a typical picture of the intestinal infantilism of Herter. When somewhat more than a year old the child had an attack of pyelitis, of which, however, she was cured and there apparently was no recurrence of this pyelitis. The child was doing well until four years later, when the mother reported that the child was not sleeping well and seemed tired, and an examination of the urine showed a considerable amount of pus. Efforts to cure this were undertaken but the task was made more difficult in this case because large doses of alkali failed to cure and hexamethylenamin caused large numbers of red blood cells to appear in the urine. The mother became discouraged and withdrew the child from treatment.

Two years later the child was brought to me on account of an injury to the face from a fall, and an examination of the urine showed still the presence of pus. The mother was then told that it was very necessary to cure this condition if possible, and that if it were not cured the child might be in danger of an acute kidney inflammation. The child was only seen five times and was again taken away.

This was in March, 1916, and nothing further was heard of the child until September, 1917, when I was summoned hurriedly to see her. The mother reported that the child had for a month seemed weak and without appetite. During this period the urine was smoky looking and contained blood. It also contained a large quantity of albumin. The child had no convulsions, but had convulsive twitchings and a blood pressure of 220.

With bowel washings, laxatives, counter-irritation over the kidneys and very small doses of hexamethylenamin the urine became clear of albumin and blood, but the child's general condition did not improve and she died a week later.

This child interested me as showing the importance of curing, if possible, every case of pyelitis, whether associated with constitutional symptoms or not, as well as the necessity for frequent examination of the urine in order to detect such cases of pyelitis when present without constitutional symptoms.

CASE OF SUPPPRESSION OF URINE FOR FOUR DAYS

The third case that I wish to report, on account of some interesting features, is that of a boy 3 years old, who, in September, 1918, while in the country, had an attack of colitis for several days, and Sept. 4, 1918, passed very little urine, and the parents became anxious. A very small specimen of urine passed was sent to the state laboratory at Albany, where it was reported to contain albumin and casts. September 5th no urine at all was passed, and the child showed edema of the face and body, and on the 6th no urine was passed. On the 7th the edema was considerably increased and there was absolutely no passage of urine. Again on the 8th no urine was passed and there was a considerable increase in the edema.

During all this period the family were assured that the child would pass urine soon, that the child would probably recover, and that when urine was passed it would contain a considerable amount of blood, which would continue for several days.

At about midnight, September 8th, the child passed urine and in the next twelve hours passed from 1½ to 2 quarts. This urine was a little cloudy, showed little sediment and on examination by cold nitric acid and heat showed no albumin, and neither blood nor casts were found. From that time on the boy gradually improved and became entirely well.

This case seemed to me especially interesting, not on account of the long period of suppression, as other cases of longer suppression have been reported, Dr. Northrup having reported one continuing for five days; but because the suppression was not associated with inflammation of the kidney, but was probably congestion or at least complete suppression of function.

211 West Fifty-Seventh Street.

DISCUSSION

DR. SMITH: I wish to put on record the case of a child who had suppression of urine for six and one-half days. This was a new-born infant who voided at birth and immediately afterward, and then had complete suppression. During this time the child had twitching, cyanosis and a high temperature, which entirely disappeared a few hours after voiding. This was a boy and a test tube was attached during the entire six and one-half days, so that we are sure the observation is correct.

DR. PISEK: I would like to ask whether that child died from nephritis or pyelonephritis.

DR. FREEMAN: No, it was simply a case of pyelonephritis. There were no casts and no edema or other evidence of nephritis.

ROENTGEN-RAY DEMONSTRATION OF ABNORMALITIES OF THE GASTRO-INTESTINAL TRACT IN CHILDREN

CHARLES GILMORE KERLEY, M.D.
NEW YORK

We have thus far made roentgen ray studies of sixty-six cases of chronic gastric intestinal disorders in children from 3 months to 15 years of age. The vast majority were between 3 and 6 years of age. Many roentgenograms were taken, in some instances as many as twenty, in order to determine the emptying time of the stomach or intestine, or both.

Case 1.—*Megacolon; dilated sigmoid.*—A boy aged 8 years, had an enormous sigmoid pouch with a capacity of at least two pints (Fig. 1). There was displacement of the liver due to the pressure exerted by the massive dilatation of the colon. The bismuth meal did not disappear entirely from the intestinal canal in seventeen days. One can readily appreciate what a difficult matter it would be to carry out any satisfactory treatment in a case of this kind without roentgen-ray examination. There was extreme abdominal distention and moderate malnutrition.

Case 2.—*Acute pylorospasm.*—This patient was a boy, 7 years of age. The roentgenogram (Fig. 2) shows that the stomach is in active peristalsis, with a pronounced pylorospasm, three hours after the ingestion of the bismuth meal. The boy had been ill for one week with extreme paroxysmal pain in the stomach to such a degree that morphin had to be administered repeatedly. It will be observed that but little of the bismuth meal had passed through the pylorus three hours after the meal was given.

Case 3.—*Constipation; elongated sigmoid.*—A boy, 6 years of age, came under observation because of obstinate constipation. There had never been an evacuation without assistance since birth. Drugs or local measures were resorted to daily. The roentgenogram (Fig. 3) of the intestine shows that the flexure of the sigmoid passes above the umbilicus and up to the transverse colon, and that the sigmoid is correspondingly elongated.

Case 4.—*Diarrhea; triple sigmoid.*—This patient was a female, 20 months old, weighing 22¾ pounds. There was a triple sigmoid (Fig. 4). During the early months of her life she suffered from obstinate constipation and about two months before coming under treatment a diarrhea developed which had continued and which it was impossible to control. There was great abdominal distention, assimilation was very defective and death followed from exhaustion. It has been found that in cases of this type a chronic mucous colitis or diarrhea is very apt to follow prolonged constipation.

Case 5.—*Recurrent vomiting; elongated sigmoid.*—This patient was a girl, aged 4 years. The stomach had not begun to empty in one and one-half hours after the bismuth meal, showing a decided pylorospasm (Figs. 5, 6 and 7). For two years the child had been a sufferer from seizures of recurrent vomiting, the

attacks lasting from two to four days. It was unusual for three months to pass without an attack. Figure 6 shows that there is retention in the stomach after five and one-half hours. Complete emptying requires about eight hours. Figure 7 shows that the child has an elongated and redundant sigmoid. There was habitual constipation and moderate abdominal distention.

CASE 6.—*Pylorospasm; angulated sigmoid.*—This was a case of marked pylorospasm. The child was a female, aged 3 years, weighing 17 pounds and 3 ounces. She had been subject to periodic vomiting attacks since birth, having a seizure about once every six weeks. There was marked malnutrition, which can readily be appreciated when one takes into consideration her age and weight. She had retention of food six hours after feeding (Fig. 8), which was corroborated by stomach tube examination. In addition to the malnutrition there was a pronounced secondary anemia. The abdominal distention was extreme. Figure 9 shows food residue in the stomach five hours and forty-eight minutes after a bismuth meal. Figure 10 represents the large intestine of the same child. There is a sharp angulation at the junction of the descending colon and sigmoid which probably accounts for the obstinate constipation and the abdominal distention. It was only through the use of enemas or drugs that an evacuation was produced. In angulations of this type it has been found that fecal matter will pass through the intestine more readily than gas.

CASE 7.—*Pylorospasm; wandering stomach; enlarged sigmoid.*—The patient, a female, 5 years of age, had pylorospasm, with the stomach displaced to the left, a so-called wandering stomach. There was a large gastric retention six hours after the bismuth meal (Fig. 11). The retention was repeatedly corroborated by stomach tube examination. The child suffered from habitually poor appetite and there was moderate malnutrition and habitual constipation. The abdomen was greatly distended. Figure 12 shows marked elongation of the sigmoid, extending two inches above the umbilicus and to the right as far as the intestine could pass.

We have found in these patients with elongated and sacculated sigmoids and resulting stasis that the emptying time of the stomach is always delayed. There seems to be direct association between the emptying time of the stomach and that of the large intestine. A peculiar feature of these cases of retention is loss of appetite. The patient will, perhaps, have a fair appetite for breakfast when the stomach is empty. The other meals must be forced or coaxed into the child.

CASE 8.—*Gastroptosis; poor appetite; malnutrition.*—The patient was a female, 16 years of age. There was marked ptosis of the stomach (Fig. 13) and an extreme prolapse of the transverse colon (Fig. 14). This patient was anemic, undersized and underweight. The only active symptom was a persistent pain in the left lower abdominal quadrant. There was no symptom referable to the stomach other than poor appetite and eructation of gas. There was moderate abdominal distention. Medication was required daily to produce an evacuation.

CASE 9.—*Gastroptosis; recurrent vomiting; malnutrition.*—This patient was a male, 11 years of age. The lower portion of the stomach is ptosed 2½ inches below the umbilicus (Fig. 15). The colon is also ptosed to such a degree that with a full bladder, the bladder, stomach and colon would come in close contact (Fig. 16). This boy suffered from periodic vomiting and had been treated for acidosis for several years. There was moderate abdominal distention and habitual constipation. The bowel function required daily supervision.

Figure 1

Figure 2

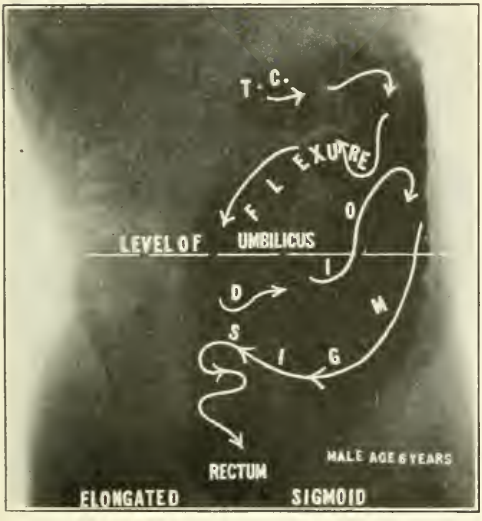


Figure 3

Figure 4

Figure 5

Figure 6

Figure 7

Figure 8

We have learned a great deal through our roentgen-ray studies. First, we have learned that there may be persistent gastro-intestinal derangements affecting the immediate and after-life of the patient, dependent entirely on mechanical agencies. The gastro-intestinal tract of the runabout child has been given the credit of being *structurally* normal, and physicians have relied on examination of the gastric contents, stools and urine with the idea of finding the causes of gastro-intestinal derangements. Our roentgen-ray studies have demonstrated that periodic or so-called recurrent vomiting, which repeatedly had been diagnosed as a manifestation of acidosis, is due, in many instances, to a dilated or ptosed stomach in which there is retention and residue long after the feeding period. Fresh food is given to these children at fairly regular intervals, in a stomach that is never empty, except in the early morning. Nature comes to the relief of the case after a time, and the stomach is emptied periodically at fairly definite intervals.

Another feature of importance relates to the appetite. We have found repeatedly that children who will not eat willingly, and who have to be coaxed and forced, may have a stomach of defective emptying capacity. A child will not be hungry in the event of food remaining in the stomach, beyond the next regular feeding period. It is our effort in these retention cases to leave a long interval between the feedings. Breakfast at 7:30 a. m., dinner at 12:30 or 1 p. m., and supper at 6 p. m. Water only is allowed between meals. With such intervals and measures employed to insure a free daily bowel evacuation, it is surprising how soon many children will develop a normal appetite.

The stomach of the normal child is emptied in from four to four and one-half hours after a meal. Food residue found five hours after a meal means a stomach of slow emptying capacity. We have further proved that there is unquestionably a relation between the emptying time of the intestine and that of the stomach. When intestinal stasis is relieved in a residue case, the stomach empties normally.

In Case 8 there was gastroptosis with surprisingly few active symptoms, other than pain, constipation and capricious appetite. Patients of this type show other symptoms, however, which are of interest. They are always undernourished. They are anemic and irritable in disposition and show faulty growth. They are ill, but the fact is not appreciated because they are as well as usual. They are the type which later in life furnishes work for the abdominal surgeon.

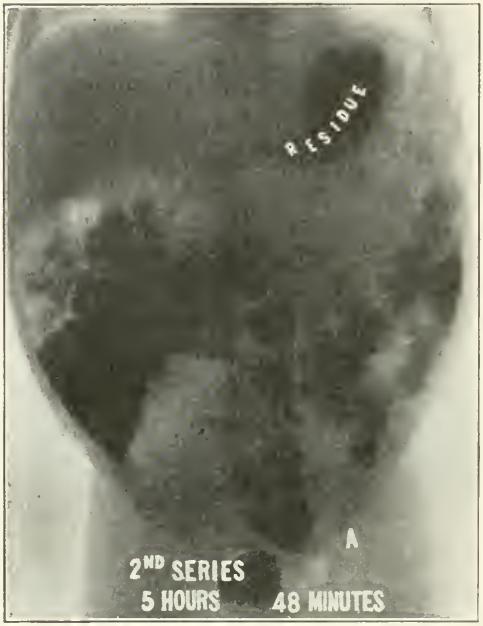


Figure 9

Figure 10

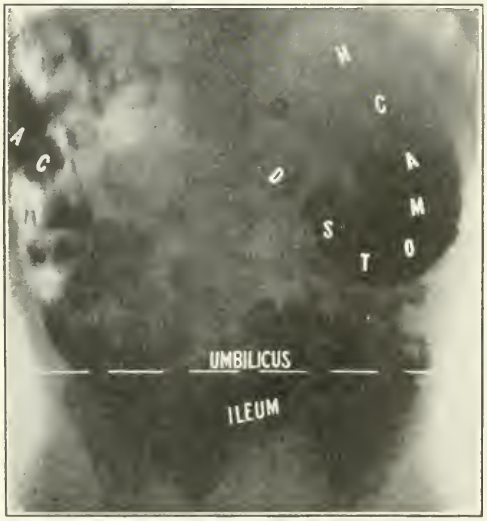


Figure 11

Figure 12

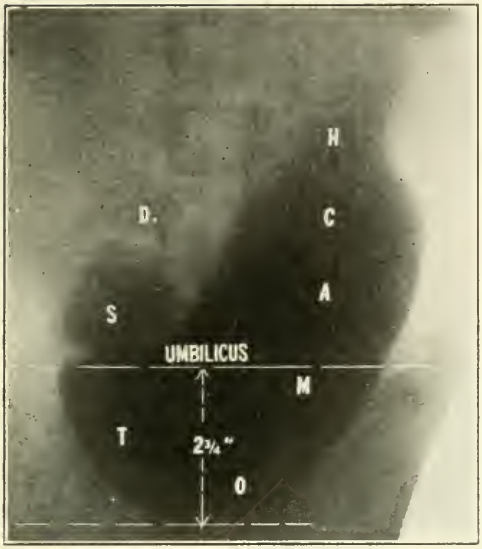


Figure 13

Figure 14

Figure 15

Figure 16

With the view to checking up our observations roentgen-ray studies have been made of children who were not ill. It has been rare to find a dilated or ptosed stomach that did not give rise to symptoms, symptoms which in some instances had not been appreciated. A ptosed colon gives trouble. Not all elongated sigmoids, however, produce symptoms. There may not even be constipation, provided there are no sacculations, adhesions or relaxed abdominal muscles.

Given a relaxed abdominal wall, a generally distended abdomen and an elongated sigmoid, we have a condition fertile in possibilities for gastro-intestinal disturbances. Weak, relaxed abdominal muscles

Figure 17

play an important rôle in these cases. When we supply adequate support, such as a tight-fitting abdominal belt, the relief furnished is very striking. The query has perhaps arisen as to what constitutes a normal sigmoid. I believe that Figure 17 represents such a case. The child was 5 years old. Figure 18 represents a normal sigmoid in a child 8 years of age.

Management.—Only general suggestions as to treatment can be made. Probably the most useful individual measure that applies to all types of abnormalities, both of the stomach and intestine, is in the use of a well adjusted abdominal belt. For those cases in which the

stomach alone is ptosed, a belt with a projecting narrow pad, a so-called shelf, is employed. The device is placed on the belt in such a position as to support the displaced stomach. In those cases in which the intestine alone is involved and in those cases in which there is a gastroptosis, an abdominal belt with a large flat pad is used. The belt is worn constantly when the patient is up and about.

Diet.—In cases of ptosed stomachs in which there is always a delayed emptying, three meals daily only are allowed, and these meals are given at as long an interval as possible. It has been found advisable to give but little fluid with the meals. After each meal, the patient rests in a recumbent position for one hour.

Figure 18

Massage.—In the cases of intestinal stasis, abdominal massage is practiced daily. It has been demonstrated that this measure is of much benefit.

Drugs. — Peristalsis has been shown to be defective when gross intestinal abnormalities exist. In addition to massage for the reestablishing of this function, certain drugs are of value. Small, frequently repeated doses of a combination of nux vomica, belladonna and cascara have been of much aid in producing a more active peristalsis and thus helping to relieve the ever present constipation. But whatever medi-

cation is selected, it should be given not less than three times a day. In cases of sacculation and dilatation of the intestine, a full dose of liquid petrolatum is given once a day. Enemas are to be used only as an emergency measure. Atropin is given in full doses when there is an evident pylorospasm.

The roentgen-ray studies on which these observations were made were carried out by Dr. L. T. LeWald, director of the roentgen-ray department at St. Luke's Hospital.

The patient is prepared the evening before by taking a cathartic and the next morning, before the injection, a cleansing soap suds enema is given. A light breakfast is allowed if desired. The opaque substance — barium sulphate — chemically prepared for roentgen-ray purposes, is mixed with fermented milk in the proportion of one part of opaque substance to six parts of the suspending medium. A sufficient quantity is used to fill the colon. This varies with the age of the patient and the size of the colon, as previously determined by roentgen-ray examination by means of the opaque meal.

The opaque meal is made up of one part of bismuth subcarbonate suspended in eight parts of the ordinary feeding mixture in infants, and for older children it is suspended in fermented milk. If the infant is unable to take the food the ordinary way, it is fed by means of a tube attached to a funnel.

DISCUSSION

DR. MCCLANAHAN: Dr. Kerley presented this subject several years ago before the American Medical Association, and since that time I have been having roentgenograms taken. I have found two cases of sacculated sigmoid flexures. I treated these cases by the injection of olive oil, massage of the bowel and small amounts of water. The series of symptoms that are revealed in these cases can be unraveled only by the roentgen ray.

DR. SHERMAN: In reference to the relation of stenosis to spasm, I would say that the greater the degree of stenosis we have the greater will be the dilatation of the stomach. All these cases of Dr. Kerley's show the stomach markedly dilated, and hence the stenosis element is prominent. In the cases I have had in which a diagnosis of pylorospasm was made the dilatation was not apt to be marked. I had one case in which the stomach was round and high up on the left side. The rounder and the higher the more is the evidence of pylorospasm.

DR. PISEK: In dilatation of the colon we may give a diet such as we give for diabetes, using mineral oils and a lack of carbohydrate. This treatment produced a cure in one instance if one can say that these cases are cured.

THE INCIDENCE AND SIGNIFICANCE OF THE RHEUMATIC NODULES IN CHILDREN

JOSEPH BRENNEMANN, M.D.
CHICAGO

The rheumatic nodules that receive so much attention from British writers on rheumatism in children are almost universally considered a rare condition with us. In all our textbooks there is a striking uniformity of expression that these nodules are common in England, but are rarely seen in this country.

Thus Holt and Howland[1] say: "They are certainly not common in this country, and although we have made it a rule to examine rheumatic patients for them, we have seen them but seldom, and they have been prominent in only eight or ten cases. This has also been the experience of most observers in this country. From published reports, however, they appear to be much more frequent in England." In the 1911 edition of Holt[2] the prominent cases had been placed at "two or three."

Koplik[3] says: "The so-called subcutaneous rheumatic nodules are seen in children less frequently in this country than in England."

Rachford[4] says: "In England these nodules are common. . . . In America, however, they are comparatively rare."

Dunn[5] says: "When present, these subcutaneous nodules are diagnostic of rheumatic fever. Judging by published reports, I should say that they are seen much more commonly in England than in this country, and probably also more often in some parts of this country than in others. They are rarely seen around Boston."

In a recent extensive paper, Murray H. Bass[6] discusses at length erythema nodosum, erythema multiforme, erythema papulatum and

1. Holt, L. Emmett, and Howland, John: Diseases of Infancy and Childhood, Ed. 7, New York, D. Appleton & Co., 1916, p. 1152.
2. Holt, L. Emmett: Diseases of Infancy and Childhood, Ed. 6, New York, D. Appleton & Co., 1911, p. 1089.
3. Koplik, Henry: Diseases of Infancy and Childhood, Ed. 3, Philadelphia, Lea & Febiger, 1910, p. 466.
4. Rachford, B. K.: Diseases of Children, New York, D. Appleton & Co., 1912, p. 403.
5. Dunn, Charles Hunter: Pediatrics, Ed. 2, 1917, **2**:329.
6. Bass, Murray H.: The Cutaneous Manifestations of Acute Rheumatic Fever in Childhood, Med. Clin. N. A. **2**:201 (July) 1918.

purpura, with only a brief mention of rheumatic nodules. Of these he says: "Strange to say this particular cutaneous manifestation of rheumatism is but rarely encountered in America, whereas it is one of the common findings in England."

About five years ago, in a paper on rheumatism in children, I expressed this same view as follows: "The rheumatic nodule is apparently a very rare condition in this country. In England, on the other hand, they seem very much more common. Thus Still finds them in nearly one half of all the cases of articular rheumatism in children under 12 years of age in the great Ormond Street Hospital wards; in nearly 30 per cent. of all the cases of chorea, cardiac and articular rheumatism in the wards, and in 10 per cent. of the naturally less serious cases in the outpatient department. We have been told by Still (personal statement) and others that we do not look hard enough, and that we expect something large while they may be very small — no larger than a pinhead. With this in mind, and looking for them constantly, I have seen only four well marked cases in this country." I had seen a fifth case with Pfaundler in Munich, and the enthusiasm with which he attacked the case amply confirmed his statement that these cases were "very rare in Germany, but that they seemed quite common in England."

Before the above paper was published some months later, I added a footnote as follows: "Since writing the above I have seen these nodules so regularly in severe cases of rheumatism with heart involvement that I am inclined to accept Still's figures for our own material from the same social stratum. Thus at the County Hospital at one time all of the children (three in number) with cardiac rheumatism had well marked nodules."

In the last five years since we have been especially interested in these nodules and have really looked for them we have become increasingly convinced that they occur in our cases quite as frequently as they do in an equal number of similar cases of rheumatism in England. There are doubtless more rheumatic nodules to be seen in London than in New York, or Chicago, or Boston, both because rheumatism is more prevalent in England and especially in "the London district"

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7. Brennemann, Joseph: Rheumatism in Children, Illinois M. J. **26**:20 (July) 1914.

(Voelcker[8]), but even more so because "it is frequently of a severe type," and it is in the severe type of rheumatic infection that the nodules attain their maximum incidence. The views that I have just expressed are fully shared by my colleague, Dr. R. A. Krost, with whom I have worked for many years in the outpatient department and in the hospital wards, where rheumatism in its severer forms is most frequently encountered.

The purpose of this paper is simply to state my observations, not with any idea of finality, but rather to attract attention to a peculiar discrepancy, in the hope that it may lead to a renewal of interest. If it will stimulate a more productive search for these nodules in this country, so that it can be established by widely distributed observers whether the rheumatic nodule is a more or less definite quantitative accompaniment of rheumatism, or whether it has a very selective geographic habitat, its purpose will have been amply fulfilled.

I am, perhaps unfortunately, not in a position to give definite statistical data as to the incidence of these nodules. Such data would, however, I believe, rather detract from the more important observation that I wish here to emphasize, that these nodules do not occur in certain stages and types of rheumatism; that they do occur fairly frequently in others, and that in certain types they are the rule.

A brief review of the early literature on this condition is of interest per se, but even more so because it illustrates how quickly we often see a large number of cases when once our attention is attracted forcibly to a phenomenon that was hitherto unknown, or considered too rare to be of interest.

The first recorded case of rheumatic nodules was that of Meynet.[9] He saw this case in 1874. The patient was a boy, 14 years of age, in whom the nodules developed while he was being treated at the hospital. Rehn[10] quotes Meynet and adds a second case in a child with recurring rheumatism. He later reported another case. Hirschsprung[11] of Copenhagen reported four cases. Troisier and Brocq[12] reported a

8. Voelcker, A. F.: Rheumatism and Chorea, Garrod, Batten and Thursfield, Diseases of Children, New York, Longmans, Greene & Co., 1913, p. 95.

9. Meynet: Lyon Med., No. 49, 1875. Quoted from Barlow and Warner.

10. Rehn: Gerhardt's Handb. **3**:21, 1878.

11. Hirschsprung: Arch. f. Kinderh. (March) 1881. Quoted from Barlow and Warner.

12. Troisier and Brocq: Rev. de méd. (April) 1881. Quoted from Barlow and Warner.

case in an adult. Barlow and Warner[13] saw their first case in 1875, and in 1881 they reported twenty-seven cases, the patients being between the ages of 4½ and 18 years. But little has been added to our knowledge of these cases since the publication of this brief but comprehensive paper. Twenty-five of the twenty-seven cases had definite symptoms of rheumatism. Twenty-six had definite cardiac involvement and the remaining case had a peculiar heart with a "murmurish first sound." Eight patients died of heart disease. Ten had chorea at the time the nodules were observed; one had had chorea some time before. They described the morphology, localization and duration of the nodules; established their pathognomonic relationship not only to rheumatism, but also to endocarditis; and pointed out on the basis of three microscopic examinations, the close structural relationship of the nodules to the vegetations on the valves in an endocarditis.

Since this time there has been a sort of stereotyped uniformity about the literature of these cases confined chiefly to textbooks. British writers like Cheedle, Poynton, Hutchinson, Thompson, Still, Cautley and others, all emphasize their relative frequency, their association with heart disease, their grave significance when abundant and recurring, their structural identity with "submiliary nodules" of the heart with some difference of opinion as to the exact histogenesis.

The status of German opinion is shown in the fact that Heubner[14] does not mention them; in Pfaundler's undisguised elation on seeing a rare case, in this instance so manifest that it could not escape detection; and in Ibrahim's[15] naive statement in Pfaundler and Schlossman (1906): "A special form of this disease, almost wholly peculiar to childhood, is rheumatismus nodosus, first described by Reynet in 1875 and popularized through Rehn and Hirschsprung. Over forty cases in children have been described up to this time (chiefly by English authors)."

Our own literature has been quoted sufficiently.

The rheumatic nodules vary in size from that of a pinhead to that of a filbert in our experience; commonly, they are the size of a split

13. Barlow and Warner: Trans. Seventh Internat. Med. Congress, London **4**:116 (Aug.) 1881.

14. Heubner: Lehrbuch f. Kinderh., 1906.

15. Ibrahim, J.: Acute Articular Rheumatism. Pfaundler and Schlossmann, Handb. der Kinderh. **1**: Part 2, 881, 1906.

pea. Doubtless they are often beyond the reach of the eye or the finger. They are always subcutaneous, never reddened or attached to the skin, and rarely tender. When the skin is loose over them there is no discolorization; if taut they appear whitish or grayish and shiny. The small ones have well been likened to a boiled sago grain. Usually they are discrete, roundish or hemispherical, with symmetrical outlines, but sometimes they are clustered, or elongated, or even radiate out in different directions. Sometimes they can be felt better than seen, but this is commonly not the case if the joint about which they occur is properly flexed, or if the skin is otherwise lightly drawn over them. On the head they must always be palpated. Over the patellae they are often better seen than felt.

They are usually distinctly movable, in fact, the smaller ones about the deeper structures of the elbow and sometimes elsewhere would often be hard to distinguish except for that fact. On the periosteal surfaces, such as the patellae and the spines of the vertebrae, they are often more firmly anchored and sometimes seem to be quite immovable.

They vary in number from one or two to a hundred or more — commonly there are eight or ten. If numerous, they are always widely scattered. The greater the number the more surely will some of them be very large, especially over the patellae, the back of the head, or on the spines of the vertebrae.

They occur always in connection with the deep fascia, the tendons, or the periosteum, but they have favorite localizations. Thus they are nearly always found, if present at all, about the condyles of the humerus, and in and about the olecranon process. Occasionally, a very large one is found near the end of the olecranon process that is almost indistinguishable till it is found to be movable. It is especially about the elbow that the joint must be flexed properly, and careful search made both with the eye and the finger. They are rarely as large here, except over the olecranon, as they are in other locations where they are connected with the periosteum. In our experience they occur relatively frequently and very large, not only along the margins as commonly reported, but over nearly the whole of the patella commonly on both knees. When the knee is flexed sharply they are very conspicuous. They are frequently found over the malleoli, especially over the external malleolus, on the styloid processes of the radius and of the ulna, but they are never large or numerous here. A striking and

not uncommon location is on the knuckles, and sometimes the tendon of nearly every knuckle is the seat of from one to five nodules. If the hand is open they quite escape notice; if the hand is tightly clenched they appear as prominent shining, whitish or grayish clusters along the tendons. Less frequently they occur on the toes.

In six or eight cases the most prominent nodules have been under the scalp, always on the back of the head. The hand should always be passed over this region. If present, there are usually from two to six, or more, large bumps nearly the size of a hazel nut.

Occasionally they show a distinct predilection for the spine, where they may be hard to find if laterally or deeply seated, and most conspicuous if located on the tips of the spinous processes. In a recent case nearly every spinous process for some distance was crowned by a large nodule and one of unusual size in the region of the first dorsal vertebra had been manipulated actively by a nonmedical practitioner in an effort to reduce a "dislocation" that was causing his "heart disease."

Occasionally they occur along the spines and vertebral borders of the scapulae, less frequently about the shoulder joint and along the crests and articulations of the ilia. I have never seen them along the tendons distant from a joint, or in the palm of the hand, or about the sternum, though they have been reported in these places.

Rheumatic nodules have a tendency to appear in groups, or waves. The life of the individual nodule varies from a few days to as many months.

It is not difficult to see why these nodules may be overlooked. One is traditionally sparing in manipulating a rheumatic patient, and especially in bending the joints. The elbows, hands and knees are commonly extended, and unless flexed they show nothing. The back of the elbow, too, is naturally turned away from the examiner in the bed patient, and it requires considerable movement to bring it into view and to palpate it properly. This is even more true of the spine of the bed patient. The spine, moreover, is naturally "bumpy." Passing the hand over the back of the head is easily omitted and in other locations the nodules must be looked for especially, and they are often hard to find. When the nodules are large and numerous they, of course, obtrude themselves.

On two points practically all observers are agreed, the rheumatic nodule means rheumatism, and it practically always means endocarditis.

Its similarity in structure with that of valvular nodules would lead one to think that both are more or less simultaneous effects of an identical cause.

In the ordinary case of articular rheumatism or of chorea, without heart involvement, the rheumatic nodule does not occur. In those cases of cardiac rheumatism in which nodules do appear they commonly follow the heart involvement after some days or weeks. Usually, only in the severe cases do they appear at the same time or very rarely a very short time before the heart is evidently affected. One of my cases, a boy, six years of age, showed on my first visit an abundance of nodules, but I could not make out a definite endocarditis until the next day. He rapidly developed a severe and protracted endocarditis and pericarditis with many nodules throughout the course of the illness. The question of how early an endocarditis is recognizable clinically would here seem pertinent.

The ordinary endocarditis that one sees for the first time in the course of an acute articular rheumatism, or of a chorea, that quickly runs a favorable course under proper treatment is usually not accompanied by the appearance of nodules. The case that at once strikes one as a severe infection, and especially one complicated by pericarditis, shows them much more frequently. The same is true of recurring and persistent cases of rheumatic infection with renewed cardiac invasions even though the individual attacks may not seem very severe. While it is true, in general, that severity and chronicity of infection are the most important factors in the production of rheumatic nodules, it is also true that nodules may occur in any case of cardiac rheumatism, and that they may be wholly absent in just those cases where experience leads one to expect them.

There is, however, a type of rheumatism in which we have nearly always found them, and usually abundantly. These are the cases in which there is severe endocarditis of long standing, often accompanied by pericarditis, where the process is active and progressive in spite of all treatment; where there is a low but nearly constant temperature; where there is wasting, with or without anasarca, and a yellowish, gray, cachectic color. These cases that sway between life and death for weeks and months, not alone because of a broken compensation, but also even more because of a smoldering, uncontrollable infection that ever and anon lights up again, are in our experience rarely free from a large number of nodules. It is in these cases that a new and

abundant crop of nodules gives an ominous warning that there is renewed invasion of a serious nature.

While the number and the size of the nodules in a general way run a parallel course with the severity of the rheumatic infection, this is not always the case; nor, from my experience, can I agree with Cheedle[16] when he says: "In the acute rheumatic fever of children when the nodules are many and large they have an ominous association with progressive endocardtis and pericarditis of the most serious kind; they indicate a grave danger, a carditis which is uncontrollable and advances almost invariably to a fatal termination." A number of our own cases that have had nodules as large and numerous as I have ever seen have ultimately, though nearly always very slowly, run a favorable course and have been left with only a chronic valvular lesion.

Though we ordinarily think of the presence of rheumatic nodules as an indication that the rheumatic infection is still active, it seems, however, often to be at least slumbering. The appearance of new nodules is here of far greater significance than the long persistence of old ones. Individual nodules will often persist for weeks and even months after all acute symptoms have subsided. We had at one time two children in the ward, each convalescing from severe cardiac rheumatism, between whom there was a very active daily rivalry accompanied with much hilarity as to which still showed the largest number and size of an old crop of nodules. In a case now under Dr. Krost's care, the child was up and about while he still had well marked nodules, though with us the rest in bed in these cases is one of months rather than of weeks.

Since rheumatic nodules are considered pathognomonic of rheumatism, it is evident that they are, at times, of considerable diagnostic value. The pathognomonic symptom is always cherished. Rheumatism in children where articular symptoms sink to a minimum and cardiac manifestatons rise to a maximum has always presented difficulties in diagnosis. Parenthetically, I might say that these difficulties are rather increased by the present tendency to make it one of a group rather than an individual and the nodules will help to maintain for rheumatism that individuality that the pediatrician will long hesitate to deny it. Several years ago I saw a child with very high fever, delirium, very stiff and painful neck, in whom I suspected a meningitis until I

16. Cheedle: Quoted by M. H. Bass.

found on more careful examination that he had a vigorous crop of nodules about the elbows. A definite endocarditis on the following day and a subsequent pericarditis confirmed the diagnostic value of the nodules in enabling me to make a diagnosis of rheumatism on the first day. Within the last week I saw a boy, 10 years of age, with a chronic infection who for two months had worn a cast over a supposed tubercular knee. He had now dullness and distant breath sounds over the left base, an endocarditis and probably a pericarditis and a very stiff and painful and rigid neck and back with a marked and persistent forward bend and with no other active joint symptoms. The presence of nodules made it more than probable that the whole process was rheumatic and not tubercular.

A peculiar interest has, to my mind, always attached to the nodules found on the knuckles. The hand is the only exposed part in this connection. By a glance at the knuckles in such a case we have been able to say: "This child has rheumatism; it is probably active; it is severe; he has an endocarditis."

Not the least value of the rheumatic nodule has been its contribution to the view that chorea is rheumatism.

A final word as to the geographic incidence of rheumatic nodules. Is it probable, as suggested by Dunn,[5] that they "are probably seen more often in some parts of this country than in others," say in Chicago as compared with Boston, or New York? A priori this seems improbable. It is difficult to see any determining climatologic or meteorologic or racial differences in the two localities. I might again refer to what I said five or six years ago which quite parallels the current statements of our textbooks. Finally, I might quote Cotton[17] who practiced pediatrics in Chicago for forty years. He says: "The fibrous nodules infrequently encountered in this country are common in England."

30 North Michigan Boulevard.

17. Cotton, A. C.: Diseases of Infancy and Childhood, 1906, Philadelphia, J. B. Lippincott & Co., p. 630.

DISCUSSION

DR. HEIMAN: About two years ago I encountered a case of this kind. I had one node excised and our pathologist, Dr. F. S. Mandelbaum, found nothing but fibrous tissue.

DR. BRENNEMANN: There has been a great deal of discussion as to the nature of these nodules. They are almost identical with the submiliary nodules in the heart.

OSTEOGENESIS IMPERFECTA CONGENITA*

H. M. McCLANAHAN, M.D., AND W. W. WILLARD, PH.G.
OMAHA

This condition was first described by Vorlik in 1849. Up to 1906, 140 cases had been reported. So many names have been used in the description of bone abnormalities in infants that an uncertainty remains as to just what is meant in some of these descriptions. For example, Lovett and Nichols,[1] after a careful study of all the case reports, conclude that only fourteen of these are true cases of osteogenesis imperfecta congenita. Since the introduction of the roentgen ray the descriptions have been more accurate. Valuable articles on the subject have also recently been contributed by Schwartz and Bass,[2] Griffith,[3] Hess,[4] Bookman[5] and Mixsell.[6]

Two types are recognized, namely: osteogenesis imperfecta congenita and osteogenesis imperfecta tarda. Authors are convinced that there is a clearly defined distinction between these two types. It is a rare systemic disease of unknown etiology, characterized by imperfect development of bone, numerous fractures and excessive callus formation.

The condition has been described in textbooks under a variety of names as follows: Osteomalacia congenita; rachitis foetalis annularis; malacia myeloplastica; periosteal dysplasia; dystrophia periostale; osteitis parenchymatosa chronica; micromelia annularis; osteoporosis congenita; fragilitas ossium.

Some authors (Griffith,[3] Schwartz and Bass[4]) uphold the theory of an hereditary tendency; others, however, find no familial influences. A study of the recent literature would indicate that syphilis is not a cause. There is no evidence of a disturbance of the endocrine glands, and environment is not a factor.

Opinion differs as to the process of bone formation in this condition. Axhausen says that there is a deficiency in periosteal bone

1. Lovett and Nichols: Brit. M. J. **2**:915, 1906.
2. Schwartz and Bass: Am. J. Dis. Child. **5**:131 (Feb.) 1913.
3. Griffith: Am. J. M. Sc. **113**: 1897.
4. Hess: Arch. Int. Med. **19**:163 (Feb.) 1917.
5. Bookman: Am. J. Dis. Child. **7**:436 (June) 1914.
6. Mixsell: Arch. Pediat. **34**:756 (Oct.) 1917.

formation due to the lowered function of the osteoblasts, but that the preparatory calcification of cartilage and the absorption of bone are normal. According to Sigowa, the pathologic process consists in a disproportion in the building up and breaking down leading to osteoporosis. Whatever the theory, the fact is that bone formation from periosteum remains in a primary fibrous state. Osteoporosis is a secondary result from the excessive formation of medullary spaces, due to deficient development of bony substance and increased absorption. In the congenital type of case, such as the one described, a more remote cause is embryonal failure of the periosteum and endosteum at the time when ossification should begin, so that the lime salts are deposited in small amounts.

The study of the bone in this case shows evidence that the osteoblasts rising from vascular fibrous bone marrow, because of insufficient nourishment or some toxic influence, did not develop properly, but remained polygonal and later underwent metaplasia into osteoclasts.

REPORT OF CASE

Baby L., male, was admitted to University of Nebraska Hospital, Oct. 1, 1918, aged 3 months. Breast fed up to the time of admission.

History.—Parents in good health. Two older children living and well. No miscarriages. Pregnancy normal and full term. Labor normal. Deformity was apparent at birth. Infant had several fractures, including forearm, thighs and legs. Masses in shafts of long bones easily felt. Head was large and soft. Cry had always been feeble; nursed readily; bowels regular and of yellow color; urine free.

Condition on Admission.—The infant lay on a cot, with thighs flexed on body, seldom moving unless disturbed. The cutaneous reflexes were normal. The abdomen was large, with an umbilical hernia. Spleen and liver were normal. Arms and legs were bent and deformed. Masses of callus could be palpated in the shafts of the humeri, femora and tibiae. Ribs felt soft and pliable, with no discernible fractures. Head felt like a membranous bag with small islands of bone here and there. The skin was soft; the eyes followed objects; no exophthalmos. Heart and lungs were normal; weight 7 pounds and 2 ounces. The mother was certain that the baby had grown, but it was not weighed at birth. Wassermann was negative; von Pirquet tuberculin test was negative. Urine: specific gravity, 1.008; albumin, negative. diacetic acid, negative; acetone, negative; indican, positive; microscopic examination negative.

Blood: Hemoglobin, 70 per cent.; red blood cells, 3,640,000; leukocytes, 18,700. Differential leukocyte count: polymorphonuclears, 56 per cent.; lymphocytes, 40 per cent.; eosinophils, 2 per cent.; special forms, 2 per cent.

As the mother did not remain with the infant, it was placed on a modified milk mixture. The child remained in the hospital from Oct. 1 to Nov. 16, 1918. During the first two weeks of its stay it made a gain of 10 ounces in weight; the next two weeks it lost 8 ounces, due to indigestion and withdrawal of food. For the next three weeks it lost 4 ounces. During the last three days

of life, while acutely ill it lost 17 ounces of weight. Ten days before death there developed numerous small furuncles over the body. A culture of the pus revealed *Staphylococcus aureus* in pure culture. While in the hospital, the temperature varied between 99 and 102 F. The bowels were normal, except on two occasions. The urine was free and did not stain. He constantly remained very quiet. The arms were placed in light splints at right angles to the body; the legs were constantly flexed. No attempt was made to apply splints to the legs. November 14, he developed a temperature of 105.6 F. with cough and dyspnea. Respirations were from 60 to 90. The temperature ranged from 102 to 105 F. during entire period of sickness. Examination of the chest revealed fine râles over the posterior chest with small areas of dulness on percussion. During the last twenty-four hours of life there were frequent

Fig. 1.—Half of transverse section of diaphysis of left femur. Decalcified in nitric acid and formol. Doubly embedded in celloiden and paraffin. Section cut 10 microns thick. Stained in hematoxylin and eosin. Photomicrograph shows an enlargement of 10 diameters. The partially ossified trabeculae are shown black. They are less numerous near the center of the shaft and extend slightly into the periosteum at the periphery. The periosteum is much thicker than normal. It reacts to fracture of the bone by still further thickening, as indicated on the left side of the section. Note the absence of a wall of compact subperiosteal bone which in the normal would extend half way to the center of the diaphysis although the total diameter of the normal is less than half that represented in this case. The blood sinuses of the marrow are the numerous smaller open spaces. Blood cells are adherent to their endothelial walls showing as a darker border to the spaces. The larger spaces in the periphery are possibly shrinkage spaces in the mucoid-like marrow. Blood forming marrow cells are abundant only in the center of the bone where they give a darker appearance to the section.

attacks of cyanosis, and for the last twelve hours of life there was a constant expiratory grunt. Death from exhaustion, November 18, with a loss in weight of 17 ounces, during this period. As far as can be ascertained from the literature, this is the youngest case with a fatal issue which has been reported in which a careful histologic study has been made.

The conclusions of Professor Willard agree very closely with those recorded by Nichols on an infant dying at the age of 10 months, the youngest previously reported.

The apparent shortening and bending of the legs are believed to be due to the pull of the muscles. The relative shortening of the legs and the long body would suggest achondroplasia. However, in the latter condition there is interference with the proliferation of cartilaginous cells in the zone between the shaft and epiphysis, resulting in shortening and premature ossification of the long bones. There is also a premature ossification of the sphenoid and the sphenoidal processes of the occipital bone which lead to skull clavis. Both of these conditions are absent, as shown by the roentgenogram and the microscopic studies of the epiphyses. The thickening of the bone is due to increased deposition of cartilaginous cells.

The soft condition of the bone, the multiple fractures and the excessive callus formation combine to make the diagnosis of osteogenesis imperfecta congenita.

REPORT ON NECROPSY

A postmortem examination was made five hours after death by Dr. J. A. Wineberg.

Inspection: Small furuncles were scattered discretely over the head, trunk and extremities. The face was that of an "old man"; the body was emaciated; weight, 5 pounds 15 ounces; the head was large, easily compressed; the arms and legs were short and curved. Callus masses were readily palpated.

Abdomen: No gross changes noted in intestines, liver, spleen or kidneys. No excess of free fluid.

Chest: Both lungs show patches of consolidation with small areas of hemorrhagic infarctions on section. Pleura shows no adhesions. Thymus and thyroid normal. Suprarenals normal. Brain not examined.

Bones: Long bones show multiple fractures with false joints in right tibia and left humerus. Periosteum not ruptured, except on the humerus and on the tibia. On removal of the bones, the left humerus was broken in being dissected out. The bones have a grayish appearance; are easily bent; are soft and pliable. A number of callus rings were present on the site of former fractures.

Microscopic Examination.—The spleen shows congestion. Large areas of necrotic tissue are present in the lungs similar to other postmortem findings in influenzal pneumonia. Kidneys show parenchymatous degeneration. Liver, thyroid, thymus and suprarenals normal. The histologic examination of the bone was made by Dr. Willard.

As the name indicates, this case is one of imperfect development of bone. The interest in the histologic side of the question centers chiefly in the attempt to discover in what way the normal process has been arrested or diverted. This is not easy to do in the absence of stages in the process, the normal of which is an extremely complex and imperfectly understood subject.

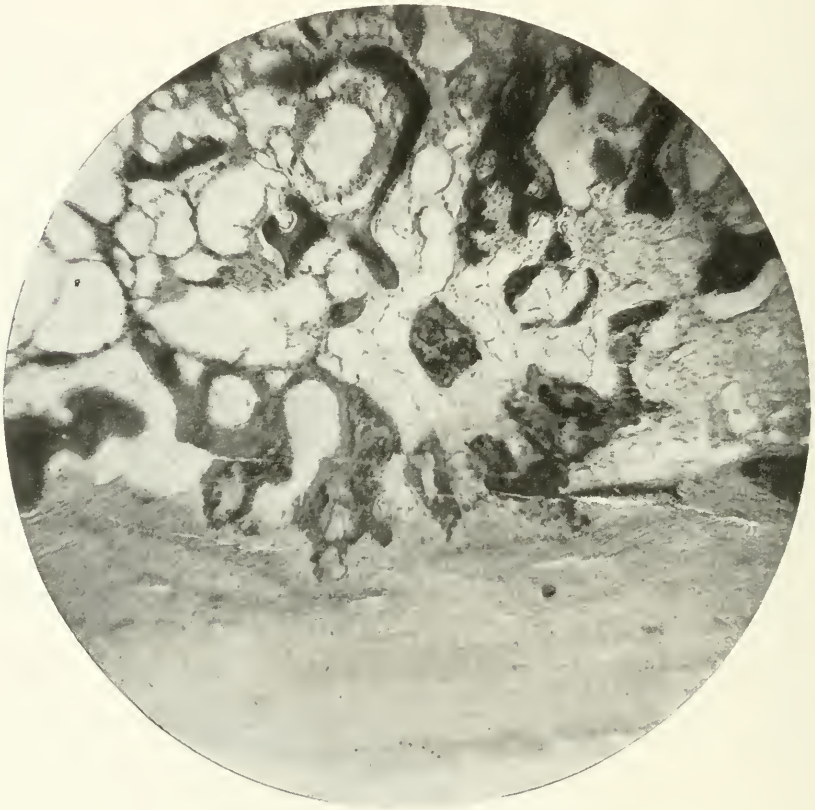

Fig. 2.—Detail from Figure 1. Magnification, 50 diameters. The full thickness of the periosteum with some adjacent bony trabeculae are shown. The darker central areas of the trabeculae are cartilage which has been formed from the periosteum.

However, the outstanding features of normal bone development are well known, involving as they do the early development of an embryonic cartilaginous skeleton which in fetal life is largely replaced

by bone and to which are added other bones developed in fibrous tissue and known embryologically as membrane bones.

The histologic structure of all bone tissue is the same regardless of origin, owing to the fact that it is always formed by accretion or deposit of osseous matrix through the combined activity of certain

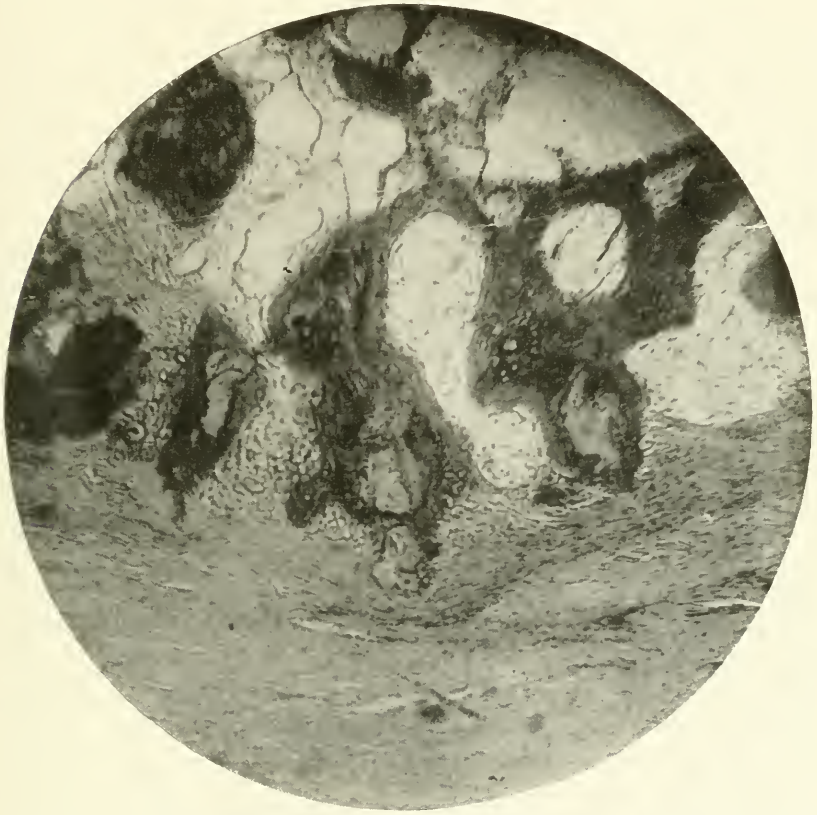

Fig. 3.—Small portion of the field shown in Figure 2. Magnification, 100 diameters. This shows the transformation of periosteal tissue into trabeculae. The encapsulated cartilage cells shown at the left represent the first stage in this process.

cells derived from embryonic connective tissue. It is thus formed locally wherever these osteoblasts are active, and when once formed, is fixed, unless absorbed. Such absorption or destruction of bone is a normal part of bone growth and is supposed to be accomplished by

large multinucleate cells known as osteoclasts. If this is true, the form and architecture of a bone depends on the balanced interplay of osteoblasts and osteoclasts. The cause behind this is unknown, and must be answered before a real explanation of osteogenesis imperfecta can be advanced.

Previous investigations on osteogenesis imperfecta indicate that all the bones of the body show much the same changes. We may, then, take the features presented by sections of the femur as typical of this condition. The sections shown in Figures 1, 2 and 3 are from the middle of the shaft or diaphysis of the bone, but they can only be fully understood through a consideration of the bone as a whole. First, consider the condition of a normal long bone in the new-born child. The shaft is slender but composed of an outer shell of compact bone, which in the case of the humerus is one fourth the total diameter. The marrow cavity is traversed by strong trabeculae entirely of bone. The cartilage of embryonic days has long since been absorbed and no new cartilage is formed. The epiphysis, on the other hand, is composed entirely of hyalin cartilage. The ossification centers that will appear later are preceded at this stage by a vascularization. The region where the cartilage abuts on the marrow cavity of the shaft is the zone of cartilage absorption and spongy bone formation. As long as the bone is capable of growing in length this zone persists. The increase in length depending directly on a process of cartilage formation in this zone between the epiphysis and the shaft of the bone. Keeping pace with this cartilage formation on the side of the epiphysis, cartilage absorption is going on next the marrow cavity. This absorption is incomplete in that slender remnants are left which offer the scaffolding on which the osteoblasts of the marrow begin the deposit of bone, thus building up bony trabeculae and contributing the necessary strength to the shaft until the sides of the shaft shall have been strengthened through the formation of a wall of compact bone laid down underneath the periosteum. With the normal growth at the ends of the bone but with the absence of periosteal bone formation at the sides, there would be formed a shaft consisting of a fibrous periosteum enclosing marrow tissue through which anastomosing bony trabeculae would run. If the actual formation of bony tissue around the trabeculae should be still further reduced, they would consist of little more than cartilage remnants with one or two lamellae of bone tissue covering them. If, along with this reduced activity on the part of

the osteoblasts or bone forming cells there exists increased or even normal activity on the part of the osteoclasts or bone absorbing cells, there is nothing left in the process to result in bone of any strength whatever.

REPORT OF HISTOLOGIC EXAMINATION OF BONE

A study of the sections in this case of osteogenesis imperfecta shows evidence that the normal processes of bone growth have been diverted in all these ways. All accounts seem to agree that cartilage is formed in the normal way and that the vacuolization of the cartilage leading to the formation of the primary marrow spaces in ossification centers occurs normally but that this process is not met by an immediate and active formation of bony tissue on the part of the osteoblasts. The cartilaginous epiphyses are normal in size and character in this specimen, but the section through the diaphysis as shown in Figure 1 shows complete absence of periosteal bone formation. The periosteum is abnormally thick but there is no bony wall underlying it. The trabeculae scattered through the marrow are slender, and section shows them to be poor in bone tissue. They all show cartilage remnants indicating that bone development has not gone beyond the initial stage. On the other hand, the actual size of the shaft is three times that of normal. Comparison shows that a normal humerus of a new-born child has a slender shaft about 5 mm. in diameter with an enlarged epiphysis of 15 mm. In this case, the shaft of the humerus has a diameter of 14 mm. while the epiphyses corresponds to that of the normal at birth. It should further be stated, that the normal shows a marrow cavity of less than 3 mm. surrounded by a wall of compact bone more than 1 mm. in thickness. The thick stocky form of the bones in osteogenesis imperfecta may be explained as the result of muscle pull on a shaft that has no supporting walls, but histologic study of the periosteum indicates that there is also a process of transformation of subperiosteal tissue into certain constituents of the marrow. This would contribute to the process that widens the marrow cavity. This subperiosteal zone is the one region where abnormal process seems to be an active one. The osteogenic layer of the periosteum, instead of forming orderly circumferential layers of bone, expresses itself in this abnormal condition through the formation of poorly developed type of cartilage. This arises in localized areas and pushing out into the marrow cavity form trabeculae which may become slightly bony as in the case of the other trabeculae. One may recognize in Figures 2 and 3 showing detail of this region groups of encapsulated cells resembling embryonic cartilage. Between the bases of these trabeculae which extend in from the periosteum multinucleate cell masses often appear. These have the structure of osteoclasts and transitional forms trace their origin to the deeper cell layer of the periosteum. Similar masses are smaller and less numerous throughout the rest of the marrow.

The marrow and its vascularization is strikingly different from the normal. The delicate reticular tissue of normal marrow is replaced by a mucoid-like ground substance containing relatively few cells. Many of these being large fibroblasts, giving tissue a distinctly embryonic appearance. Throughout the marrow are large sinusoidal blood vessels with distinct endothelial lining. The characteristic marrow cells of blood-forming function were found in abundance only in the central part of the section. Careful analysis of these cells could not be made from the material thus far studied, but typical myelocytes, nucleated red cells and megakaryocytes (giant marrow cells) were prominent.

In the foregoing general account there has been no attempt to discuss the literature bearing on the microscopic study of the bones in cases of osteogenesis imperfecta. This will be left to a later study of the cellular elements. This case, as far as presented, agrees in all essentials with the description by Michel and others.

DISCUSSION

Dr. Brennemann: We ordinarily think of these cases as extraordinarily severe. I had one child who was in perfect health. It had some kind of a nutritional disturbance, but was not very sick. Its arm then became sore and it had a flail joint and a few days later the other arm became sore and all four bones of the forearms were broken. We had a roentgenogram taken and thought we would find a green stick fracture, but found that the bones were broken absolutely straight across. They healed rapidly and with unusually distinct calluses and the child seemed to be normal otherwise. The only thing is that these children worry one because one is always over a volcano and is always at the mercy of any slight trauma.

Dr. Holt: I saw recently a child 11 months old who had a spontaneous fracture at the middle of the tibia. Scurvy was first suspected because of swelling and tenderness. Six months later a similar fracture occurred on the opposite side; both were transverse fractures without displacement. The roentgen ray also showed that in the interim a third fracture had occurred in the tibia that had not been recognized by symptoms. In a second case coming into the hospital a few months ago a diagnosis of scurvy was also made because of acute tenderness and swelling. The roentgen ray in that case also showed a typical fracture.

In the first mentioned case the mother gave a history of frequent fractures during childhood, from the fourth or fifth year to the fifteenth year. Any slight trauma produced one. Apparently the influence of heredity is of some importance.

Dr. Herrman: As to heredity, only about one half of these cases show a family history of fragility of the bones, so that it is probable that there is a hereditary factor. It is not only the bones that are abnormal, but also the ligaments and other structures. The abnormality is not limited to the bones themselves. Another feature is the frequent association with a blue color of the sclera, which is present in about one half of the cases, and that, too, indicates that osteogenesis imperfecta is not a disease confined to the bones, but probably many of the other tissues of the body are somewhat abnormal.

Dr. McClanahan: I should have added that the Wassermann test was negative and the von Pirquet was negative. The blood showed a secondary anemia.

As to heredity, the most that is said on that subject is by Griffith in the *Journal of Medical Sciences*. He states that 25 per cent. give a family history of the condition. Nichols and Lovett in 1906, collected 140 cases and on careful study they found that only fourteen of these could be considered true cases of osteogenesis imperfecta. From a careful study of these cases we think that there are two types of cases, osteogenesis imperfecta congenita and osteogenesis imperfecta tarda. In the congenital type fractures may be found at birth which cannot be attributed to difficult labor.

CARE AND TREATMENT OF WHOOPING COUGH PATIENTS

JOSEPH E. WINTERS, M.D.
NEW YORK

In the five-year period, 1911 to 1915 inclusive, whooping cough caused 835 more deaths than scarlet fever in New York state. In 1916 whooping cough caused almost as many deaths as typhoid fever and scarlet fever combined in New York state. In 1917 whooping cough caused 140 more deaths than typhoid fever and scarlet fever combined in New York City. In 1918 whooping cough caused almost twice as many deaths as typhoid fever and scarlet fever combined in new York City. In whooping cough cases, 96 per cent. of deaths were in children under 5. Of these, only 2.5 per cent. were in the fifth year. Three fourths of whooping cough deaths are in children under 2, two thirds in children under 1 year of age. The mortality of whooping cough is 50 per cent. higher in the first year than in the second.

During the first year of life whooping cough causes more deaths than any other infectious disease. In the same period, susceptibility to it is greater than that to any other infectious disease. The younger the child the greater the susceptibility and the higher the mortality. This extreme susceptibility and high fatality make prevention paramount.

PREVENTION

Whooping cough infection can be carried only in clothing infected by the coughing of the whooping cough patient. The first year child cannot walk to infection. He is taken to it by a responsible adult, or it is carried to him in infected, soiled clothing. The carelessness of the attendant, or the neglect of the physician to disseminate preventive information, should be inscribed on the whooping cough death-column.

Prevention in childhood is permanent prevention. Waning susceptibility obliterates whooping cough. Age immunity is usually attained at the end of the tenth year, and it is practically assured after puberty.

Two sisters, 16 and 18, were said to have whooping cough. Calomel, salines, correct diet to cleanse the alimentary canal and secure a clean tongue stopped the cough.

A gentleman whose three grandsons were under treatment for whooping cough said he had the disease. Purgatives and rigid diet for three days eliminated the cough.

The same nerve supplies the respiratory and the digestive organs. To denominate a persistent reflex cough whooping cough is a common error. For forty years, every case of adult cough denominated whooping cough has proved to be a digestive reflex cough.

After the tenth year, a diagnosis of whooping cough must not be affirmed until the digestive tract has been effectually cleansed throughout from above downward and a rigid diet maintained for two weeks to prevent the probability of digestive reflex cough being erroneously denominated whooping cough. Quarantine should be instituted during this period. Age immunity makes childhood prevention an imperative duty.

TREATMENT AND REGIMEN

Whooping cough infection does not kill. It is the complications which kill. Whooping cough infection does not cause the complications. They are caused by bad management—a reproach to those who execute or those who prescribe the management. The object of treatment is to obviate complications, to keep the disease simple, to keep it mere whooping cough. Two thirds of the deaths are caused by bronchopneumonia. Other causes are convulsions, meningitis, diarrhea and vomiting and marasmus. Bronchopneumonia as a complication occurs in winter, others chiefly in summer.

The summer suspension of bronchopneumonia is radiant revelation of the cause. Nature's teachings are the physician's guide. In winter, the whooping cough patient must have summer temperature.

Rest prevents complications. There are from twenty to forty or more attacks in twenty-four hours. In the explosive, expiratory cough, the heart is compressed and emptied, the veins are engorged, the pulse is rapid, weak, irregular and dicrotic. In the long-drawn, crowing inspiration, there is dilatation of the right heart and congestion of lungs. Twenty or more times in twenty-four hours there is dilatation of the right heart, congestion of the lungs, compression of the heart, engorgement of the veins, and rapid, weak, irregular, dicrotic pulse. When there are forty or more attacks in twenty-four hours, the child will probably die of some of the complications.

The attacks are excited by exercise, excitement, emotion (crying, screaming), a strong current of air, changing from a recumbent to an

upright position and by distention of the stomach. Fatal complications may be obviated by rest, warmth and by food adapted to age, digestive ability, season, and constitutional condition. The patient should be kept indoors in an equable temperature. The room should be maintained at 65 F. Fresh air should be supplied but no possible draught should be allowed on patient. The bedroom in which the child sleeps must not be colder than that in which the day is passed. Violation of these rules induces attacks; repeated attacks superinduce complications.

In the Evelina Hospital for whooping cough, the children were worse with every change in temperature, every windy, stormy day. The disease runs a much less determined and persistent course in summer than in winter. The character of whooping cough in summer, and when the child is shielded from exposure, reveals to us that we must lessen the number, shorten the duration, and mitigate the severity of attacks.

The Pharmacopeia contains a safe drug for lessening the number, shortening the duration and mitigating the severity of the attacks. The drug is codein. For a child 1 year old:

Codein, 1 grain; water 3 ounces. Teaspoonful every four hours for three doses. Then discontinued for twelve hours. Future dosage to be determined by the effect.

For a child 2 years old:

Codein, 1 grain; water, 2 ounces. Teaspoonful every four hours for three doses. Then discontinued for twelve hours. Future dosage to be determined by the effect.

For a child 3 years old:

Codein, 1 grain; water 1½ ounces. Teaspoonful every four hours for three doses. Discontinued for twelve hours. Subsequent dose according to the effect.

The chest of whooping cough patients should be examined in the interval of attacks. There should be normal physical signs, no râles, no bronchitis, and normal respiratory murmur.

Lessening the number, shortening the duration, and mitigating the severity of the attacks — of congestion of lungs — wards off bronchitis. Repeated attacks of bronchitis for from six to ten weeks causes infiltration into, and enlargement of, the tracheobronchial glands. This may occur at any remote period of life. Rest, warmth and sedative treatment obviate these dreaded sequela of whooping cough.

This treatment makes whooping cough a complication-free disease; it is the complications which kill. With the correct management of the whooping cough patient, the discreditable mortality column of whooping cough will be effaced.

The following from the *American Journal of Diseases of Children* for July, 1918, pages 23 and 29, is conclusive as to the futility of pertussis vaccine. The report is from the Home of Hebrew Infants, New York City (the first in 1914 by Dr. Hess; the second for the past year by Dr. Barenberg):

25 West Thirty-Seventh Street.

The vaccine did not seem to be of value in curing the disease or lessening is severity. Pertussis vaccine, given even in large dosage, not only has no curative effect, but does not tend to lessen the severity of the disease. The experiences of these two epidemics can be interpreted only in this way.

DISCUSSION

DR. ADAMS: A subject as important as this should not go without discussion. The paper contains certain aphorisms that I do not believe all can accept. Many of the statements made are entirely at variance with what some of us believe and practice and therefore we should not allow the paper to go without discussion. It is not necessary to give a dissertation on the subject; what we want to know is how to prevent and how to cure whooping cough. Some years ago we had a symposium in this society on the different methods of treating whooping cough. Men reported having treated whooping cough in different climates. Some had treated patients at the seashore and some in the mountains; some in the South, and some in the central states. The consensus of opinion was that the child with whooping cough should have good food and fresh air and should otherwise be left alone so far as treatment is concerned. Many did well at the seashore and many did as well at the mountains. The men agreed that no specific principles of treatment could be established.

The method of spreading the infection as laid down by the writer is unique. I do not know that we can accept the statement that infection must be taken to the child in clothing. I can conceive that if a child with whooping cough vomits and the vomitus gets on the clothing of the parent or attendant and a susceptible child comes in contact with that clothing it might contract whooping cough. There must be some close contact in order to convey the disease from the patient to another person. I can conceive that if you pen a child up in a close room or in school with another child who has whooping cough the disease may be conveyed to the susceptible child.

As to treatment by vaccines, in the literature we find a variety of opinions which do not coincide. I think vaccines have no effect either in preventing the child from having the disease or in stopping the paroxysms. Some children get along without much treatment and others require sedatives. Others, again go out in the open air in winter or in summer and do well.

Some years ago I made a diagnosis of whooping cough in an infant belonging to a man in the government service. He then took the infant up into the mountains in a lonely camp. When he returned he reported to me that

he had had an awful time but had cured the child with vaccines. I said that I had not had any experience with vaccines and would like to know how long it took to effect a cure with them. He replied that the child got well after two months. I told him that any case ought to be better after two months without any treatment. Subsequently this cure by means of vaccines became something of a joke.

Now, Dr. Winters has brought up the question of whooping cough in adults. A digestive cough should not give any trouble in diagnosis; of course in laryngismus stridulus there might be a mistake, but certainly there is no reason for making a mistake in differentiating between a digestive reflex cough and the paroxysmal cough of whooping cough.

On one occasion I wanted to demonstrate the paroxysmal cough of whooping cough to a class, and I gave the child crackers and water and tried in other ways to get him to cough, but without success. An intern succeeded in bringing on a paroxysm. I asked how he had done it, and he said he had given the child a hard pinch.

DO CALORIES MEASURE THE VALUE OF FOOD?*

HENRY DWIGHT CHAPIN, M.D.
NEW YORK

As the attention of physicians is being called more and more to problems of nutrition and their solution, it may not be out of place to reexamine some of the foundations of our teachings and see if they are altogether sound.

CALORIES AND FOOD VALUES

Much is written and said about calories, as, the food value of a certain substance is so many calories; and there is a suggestion that a calory is a sort of food ingredient, the real value of food depending on the number of calories present.

A calory is a measure of heat, just as grams, ounces or pounds are measures of weight. If a table of food values was prepared which stated that a definite quantity of milk yielded 6 pounds, meat 8 pounds, bread 4 pounds, and soup 1 pound, the natural question would be, "Pounds of what?" Scant attention would be paid to any one dwelling on the value of particular foods, because the pounds were there. Is it any more rational always to dwell on the value of a definite quantity of food simply because it contains so many calories? The most diverse kinds of food may have the same caloric value but a very different nutritive value.

The calory method of feeding is based on the assumption that nutrition processes depend solely on the oxidation of food, and that the heat given off as the result of the oxidation is the sole measure of the value of the food. In practice, the tendency is to keep a table of calory values from which a diet is made out by an arithmetical process, and the physician is likely to feel that if the calory count is correct, the food problem is properly solved.

In experimental nutrition, however, it is soon found that nutrition is not a simple oxidation process. Many feeding experiments have been conducted with animals to see if food materials from different sources, having supposedly the same chemical composition and yielding the same number of calories as the result of oxidation, have the same practical food value. One of the most notable of these experiments

was made by Hart, McCollum, Steenbock and Humphrey[1] at the Wisconsin Agricultural Experiment Station with a large number of animals over a period of four years, and some of their remarks and conclusions make interesting reading. They say, in part:

> There is evidence from the data that there is a distinct and important physiologic value to a ration not measurable by present chemical methods or dependent on the mere supply of available energy. While the latter are important and give valuable data for a starting point, they are, nevertheless, inadequate as final criteria of the nutritive value of food. . . . Probably none have felt the limitation of mathematically constructed feeding standards more than those who have taken a prominent part in their development, and even the practical and successful feeder uses these standards only as a help, varying the kind, as well as the proportion of total nutrients in the ration to meet the requirements of the individual. The kind of nutrients, however, receives his attention only when their effects are extremely pronounced and immediately apparent. . . . But in addition to the limitation of mathematical standards, which consider only digestible nutrients, or the total net available energy of a ration, there are still other important factors that must be considered. We refer to what may be called the *physiologic value* of the ration. . . . There are many different proteins, in addition to nitrogen-bearing bodies of nonprotein character; fats of different compositions and degree of saturation; carbohydrates of many types; and almost a host of undetermined and undefined bodies in the daily ration of a domestic animal. . . . Unquestionably the *physiologic* value of a ration is largely dependent on its chemical constituents, but the usual determinations made on feeding materials do not reveal the character or manner of combination of many of the constituents. Consequently, the physiologic value can be determined in the present state of our knowledge only by long continued observations of the reaction of the feed on the animal.

Since the foregoing experiments were conducted, animal feeding experiments have been widely carried on, and it seems to be the universal finding that the number of calories a food yields on oxidation is not at all an indication of its feeding value. Bayliss[2] says:

> Heats of combustion do not necessarily give the actual energy value of foodstuffs as available in the organism. . . . In the animal body, energy is derived from chemical combination. This form of energy is readily converted into various other forms without the necessity of passing through the form of heat.

ANAEROBIC AND AEROBIC REACTIONS

At this point it may be well to call attention to a fact not generally known, namely, that large quantities of the common foods are used

1. Research Bull. 17, University of Wisconsin Agricultural Experiment Station.
2. Bayliss, W. M.: Principles of General Physiology, New York, Longmans, Green & Co., 1915.

by animals by anaerobic metabolism, since air is not essential to animal life in all cases. Mammals and birds quickly die from lack of oxygen. Frogs may remain alive for days in an atmosphere containing no oxygen. Fish and reptiles may live without air for some time, while insects will live and be active in a vacuum for several days. Leeches can live ten days without oxygen. Bunge[3] says:

> I have made many experiments with the roundworm of the cat, and have satisfied myself that these animals can live in media entirely free from oxygen for from four to five days and be extremely active during the whole time. Whoever has seen these movements must be convinced that oxidation is not the source of muscular energy in these animals.

It is evident that animals which eat the common foodstuffs and obtain energy from them for days at a time in the absence of oxygen do not carry on their vital processes and obtain energy as the result of oxidation, and that for them the amount of heat that can be produced by oxidation of the food they utilize is of no interest.

In the bodies of higher animals it appears that the processes of nutrition are a combination of anaerobic and aerobic reactions. From reading Bayliss it would seem to be fairly well settled that the primary processes in the bodies of higher animals are anaerobic, and that the aerobic processes are secondary. The contraction of the muscles is an anaerobic process:

> There is neither consumption of oxygen nor evolution of carbon dioxid. It is not an oxidation process. . . . To restore the potential energy which the system has lost in contracting, energy is supplied by another reaction of a chemical nature, which succeeds the contractile stage. . . . The energy required for the second process is afforded by a reaction in which some substance, carbohydrate or fat, is oxidized. Much oxygen is used and carbon dioxid given off. . . . Consideration of life without oxygen leads to the view that the actual source of the free energy required by a living organism is a secondary matter. If it cannot be obtained by oxidation, other chemical reactions, although of a less efficient kind, are made use of.

If this is the case, it is easy to understand how the large number of animals that can live without oxygen obtain their energy—they simply use more food and discard it at the point at which oxidation begins in the aerobic animals.

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3. Von Bunge, Gustave: Textbook of Physiological and Pathological Chemistry, Philadelphia, P. Blakiston's Son & Co., 1902.

VALUE OF THESE FACTS TO THE PHYSICIAN

These facts are of much importance to the practicing physician. He is required to solve numerous problems which call for the selection of food of the right *physiologic value* for a given individual. What may be the right physiologic value for one may not be suitable for another. It is possible that the difficulty lies in a perversion of anaerobic metabolism in one case and aerobic metabolism in another. It has been conclusively shown that in practice the caloric value of a food is no strict indication of its nutritive value, but it has not been shown why this is so. With the recognition of the fact that an animal body is not merely a furnace in which food is burned, but that a long series of chemical changes in the food takes place, it is not difficult to see why practical results often cannot be obtained with foods valued only by their oxidation properties. To quote Bayliss again:

> The general conclusion seems to be justified that the cell mechanisms are such as to be able to use chemical energy whether it comes from oxidation or otherwise, and that they are independent of the particular chemical reaction which affords it.

The animal body is highly complex, with a metabolism that is in part anaerobic and in part aerobic, as some tissues live by the former process and others by the latter.

SUMMARY

Heat or energy may be produced by chemical cleavage as well as by oxidation. Heat may be a degradation of energy, and in the human organism it is an excretion. Heat measurement alone is not a safe guide for the calculation of food values. This is especially true at the beginning of life when growth is the all-important factor. The foods that *build* rather than those that readily undergo oxidation must be properly gaged if we are to have healthy development.

A false theory does harm in that it points in the wrong direction, and it is a question whether it is not time to weigh carefully the calory theory of feeding and restrict or partially abandon its use. To take its place, a system of teaching must be constructed based on a knowledge of the physiologic properties of the various food elements and the fact that there are mixed types of metabolism. Some form of biologic testing of foods must be elaborated if an always reliable gage of nutrition is to be established.

51 West Fifty-First Street.

DISCUSSION

DR. TALBOT: I am sorry to disagree with Dr. Chapin's conclusion. Of the men I know he is the last one with whom I should wish to disagree. But in the first place all physiologists agree that conclusions from observations on cold blooded animals cannot be applied in all instances to living warm blooded children. He has compared a condition which is not compatible with human life—the human infant. It has been conclusively shown in the Carnegie Laboratory that every time a muscle moves it increases the production of carbon dioxid, and carbon dioxid cannot form without the necessary oxygen to combine with the elements which come from the changes which take place during muscular contraction.

I think the trouble with the caloric feeding in the past has been lack of knowledge of the foundation on which it is based. I do not mean to say, however, that the use of the caloric method is the only method of feeding, but I do mean that the assumption that we are using an accurate method is very inaccurate. When we feed an ounce of whole milk we assume that 21 calories are being fed. That is very inaccurate, because the composition of milk is known to vary a great deal. I do not maintain that paying attention to the caloric estimation is the way to feed a baby. We must first feed the baby what agrees with it and what it can digest and in the second place we must see that the food is well balanced. In the third place we must see that the baby gets enough and when the normal healthy baby gets enough it will be getting what the physiologist shows is the proper number of calories.

DR. SEDGWICK: Like Dr. Talbot, there is no one I dislike more to disagree with than with Dr. Chapin. I will say first that one of the first articles I ever published was on this subject. It was very crude and was published where you will probably never see it and of that I am glad, but I am still of the same opinion that I was at that time. I felt at that time and still feel that the consideration of the caloric value of food is a very valuable aid in determining the quantity of food. That was what I felt in 1905 or 1906, and I still feel the same. The estimation of the caloric value of the food is a great aid but it is not a method of feeding. One could feed protein, or sugar or fat completely and get a sufficient number of calories, hence it is very bad to use the term "caloric method of infant feeding." I am a mere tyro in this subject compared to Dr. Talbot, as most of us are, and I am glad to hear that he believes the same as I do on this subject.

DR. SCHLUTZ: I possibly misunderstood Dr. Chapin, but he stated, I believe, that oxidation had nothing to do with metabolism. According to the knowledge of biochemists practically all the changes involved in the metabolism of food finally resolve themselves in either oxidation or hydrolysis. Whether the change is brought about by aerobic or anaerobic agencies is not so essential as is the fact that the final fundamental chemical process is either oxidation or hydrolysis.

I feel as both Dr. Talbot and Dr. Sedgwick do that the caloric measure of food is something we can not well discard. It is the only accurate means we have of measuring potential energy production of food. It should, of course, be generally understood that it is merely a mathematical standard of measure. One can hardly speak of it as a system of feeding.

DR. MARRIOTT: In considering such a problem two well established laws must always be taken into consideration; the law of the conservation of matter, and the law of the conservation of energy.

Ingested food is either excreted unchanged, oxidized to supply energy, or stored in the body. That the energy is produced by oxidative processes is proved by the fact that the end-products excreted are oxidation products, and further by the fact that the results of direct and indirect calorimetry agree; that is to say, the heat actually produced by the body is the same as the heat calculated on the basis of oxygen consumed. Mechanical energy is, of course, ultimately dissipated as heat and may be measured as such.

Dr. Chapin's frog is no exception to the rule; he does not live without oxygen any more than any other animal. The frog, being a cold blooded animal and remaining at absolute rest during the winter, requires very little fuel and produces very little heat, but what heat is produced is quite proportional to the oxygen consumed. He can live for a long while on oxygen stored in the body either as such or in the form of foods relatively rich in oxygen, for example, carbohydrates, which when converted into fats give an available supply of oxygen.

All foods are not suitable for storing; for example, proteins deficient in certain essential amino-acids cannot be laid down as body protein, no matter how much is fed or how great the caloric value.

The body requires a certain amount of fuel to supply energy, and this fuel must be supplied by the food or else it comes from the body tissues themselves. When food in excess of the energy requirement is taken in, absorbed and retained, the excess is necessarily stored in some form and there is a gain in weight. That is simple mathematics.

The failure of the so-called caloric method of feeding is not because calories do not measure food value, or that energy can be produced without oxidation, or without anything else, it is simply because we never know exactly how many calories any individual infant may need in order to grow. He may need 80 calories per kilo or he may need 180. We often make the mistake of assuming arbitrarily that an infant needs so many calories and when he does not gain we jump to the conclusion that a calorie is not a calorie, or that the theory of nutrition is wrong or that energy can be produced out of nothing, when as a matter of fact the only thing wrong is our assumption. The calories, the baby and the theory of nutrition are all correct.

DR. HOLT: I am glad the caloric method has been brought up for discussion simply to say that it is not to be regarded as a method of feeding. Dr. Rotch introduced percentage feeding and it came to be known by the misnomer of "a method of feeding," so the caloric measure of the food has come to be known as a method. The essential idea of percentage feeding was exceedingly important and I greatly regret that it has been lost sight of. We have dropped something of great value when we cease to employ it. It is of the utmost importance that we should know not only how many calories a child is getting but also what proportion is fat, what part carbohydrate and what part protein. The percentage method simply states what these proportions are. The caloric estimation is also of value but it should be looked on only as a method of statement of the energy value of the food given, not as a method of feeding.

DR. CHAPIN: I feel very much rewarded by the discussion that has followed the reading of my paper. I would thoroughly agree with most points that Dr. Talbot brought up, but not the first in which he stated that observations on lower animals were not to be considered in connection with problems of nutrition in human beings. One great trouble we have had is that

we have detached ourselves too much from science at large. One cell lives and breathes and grows just as a higher cellular animal. Many errors have been made because the physician has detached the higher from the lower in animal life. Evolution starts with the living cell and goes up, and I would like to take exception to such a statement because it is constantly brought out in medical meetings.

I thoroughly agree as to the inaccuracy of this method alone. My reason for bringing it up is not especially for the members of this society but because all over the country there are men with little pads writing out calories and they feel that the final solution of infant feeding has been reached. Dr. Holt has emphasized the advantages of knowing the proportions of the food elements, but when it comes to calories, we can get calories from almost anything, coal, gas, meat, cereal; they all produce heat, but that they are interchangeable is absurd. If that point is clarified, we have gained something. I have simply taken the recent things that Bayliss, MacCollum and others have brought out, and the important point is that many processes of life take place without oxidation, and if we accept that fact it throws out a great deal of value in the estimation of calories.

STUDY OF THE STOOLS IN CHILDREN'S INSTITUTIONS SHOWING THE INCIDENCE OF INTESTINAL PARASITIC INFECTIONS

L. R. DE BUYS, B.S., M.D.
Professor of Pediatrics, School of Medicine, Tulane University
NEW ORLEANS, LA.

AND

H. L. DWYER, M.D.
KANSAS CITY, MO.

This study consisted in the examination of the stools of 595 individuals in seven institutions. The ages of the inmates varied from three months to eighteen years and included institutions in which there were both males and females.

The method of examination was as follows: The stools were secured at the various institutions and placed in pill boxes, and each box was labeled with the name of the individual from whom it was obtained. The stool was thoroughly macerated in distilled water and filtered through several thicknesses of gauze. The filtrate was then placed in a centrifuge with a velocity of 1,500 revolutions per minute for from ten to twelve seconds, after which the supernatant fluid was discarded, the residue again thoroughly mixed with distilled water and again centrifuged for a like period. If the fluid after the second centrifuging continued cloudy the residue was again washed and centrifuged a third time. Then the water was poured off and the residue spread on a slide. The examination was made with a minimum of light. This precaution was taken so as not to miss *Hymenolepis nana*, which is very translucent. The preparation was made thin enough for newspaper print to be discernible through it.

The specimen was then placed under the microscope and examined with the lower power to find the ova and then with the higher power for closer study and differentiation.

The time required for the examination of a specimen varies considerably depending on the severity of the infection and the character of the stool. Stools from purges are more difficult to examine. If watery, the preparation is cloudy and if there is much mucus it probably interferes with the ova being thrown to the bottom of the test tube.

In this series no stool was considered negative until about one-half hour's search failed to reveal anything. Cercomonas and trichomonas were found in about fifteen instances in the stools examined.

Observations were made on the test tubes in which heavily infected specimens were contained to determine the possibility of ova being carried over in subsequent examinations and increasing the positive findings. It was shown, however, that after being washed and boiled the findings were negative on repeated examinations. This dispelled the possibility of the ova adhering to the sides of the tubes being hard to remove. No tubes were used twice on the same day.

In making the examinations of blood the stool findings were not kept in mind, the counting being done independently.

The stools were secured from seven institutions, which included four orphan asylums, one infant asylum and two waif's homes. The number of inmates in the institutions were, respectively, 95, 125, 75, 67, 110, 51 and 72; a total of 595.

Of the 595 individuals whose stools were examined, 317, or 53.27 per cent., were infected. Of the 595, 211, or 35.47 per cent., had single infections; eighty-two, 13.8 per cent., had double infections; twenty-one, or 3.53 per cent., had triple infections, and three, or 0.5 per cent., had quadruple infections.

Two hundred and nine, or 35.1 per cent., of these individuals were infected with *Trichuris trichiura;* eighty-six, or 14.62 per cent., were infected with *Ascaris lumbricoides;* forty-one, or 6.89 per cent., with *Oxyuris vermicularis;* forty, or 6.72 per cent., with *Strongyloides stercoralis;* eighteen, or 3 per cent., with *Necatur americanus;* fifty-five or 9.25 per cent., with *Hymenolepis nana,* and one, or 0.17 per cent., with *Hymenolepis diminuta.*

Of the 317 instances of infections, 211, or 66.56 per cent., were single; eighty-two, or 25.87 per cent., were double; twenty-one, or 6.62 per cent., were triple, and three, or 0.95 per cent., were quadruple.

As to the type of infection in the 317 instances: *Trichuris trichiura* was offender in 209, or 65.9 per cent.; *Ascaris lumbricoides* in eighty-six, or 27.13 per cent.; *Oxyuris vermicularis* in forty-one, or 12.93 per cent.; *Strongyloides stercoralis* in forty, or 12.62 per cent.; *Necator americanus* in eighteen, or 5.68 per cent.; *Hymenolepis nana* in fifty-five, or 17.35 per cent., and *Hymenolepis diminuta* in one, or 0.32 per cent.

THE INSTITUTIONS

The orphan asylum designated in the tables as No. 1 is an institution in which the most complete data could be obtained. Ninety-five individuals were examined, forty-six of whom were males and forty-nine females. Seventeen showed infections. Their ages ranged from 1½ years to 18 years. The previous environment of the inmates was usually known. In seventy-five instances it was unsanitary; and in six sanitary; in fourteen instances the previous environment was unknown. The duration of the residence in this institution of those studied ranged from a short period up to twelve years—in the infected cases from six months to ten years.

In the seventeen infected cases in this institution, fifteen were of those whose previous environments were unsanitary, one sanitary and one unknown. The parasitic infection of the seventeen in the group were *Trichuris trichiura* in ten, or 58.82 per cent., of the instances; *Oxyuris vermicularis* in six, or 35.29 per cent.; *Hymenolepis nana* in two, or 11.76 per cent., and *Ascaris lumbricoides* in one, or 5.88 per cent., of the instances. The double infections were one each of *Trichuris trichiura* and *Ascaris lumbricoides*, and *Trichuris trichiura* and *Hymenolepis nana*.

There were no animals in the institution. Raw washed fruit and vegetables were eaten. A dietitian is in charge of the institution. There were among the inmates no crawlers, finger suckers or dirt eaters. The stools had been examined once in the past six years. A systematic medical examination is made about twice annually.

In the orphan asylum designated in the tables as No. 2, 125 individuals were examined, 101, or 80.08 per cent., of whom were infected. There were forty-three males and eighty-two females. Of those infected, forty-three were males and fifty-eight females. It is interesting to note that all the boys showed infections. The ages of the inmates ranged from two to seventeen years. The duration of residence in the institution was unknown. The infections in the 101 cases were as follows: *Trichuris trichiura*, seventy-three, or 72.29 per cent.; *Ascaris lumbricoides*, twenty-six, or 25.34 per cent.; *Oxyuris vermicularis*, nine or 8.91 per cent.; *Strongyloides stercoralis*, twenty-one, or 20.89 per cent.; *Necator americanus*, eight, or 7.92 per cent., and *Hymenolepsis nana*, fourteen, or 13.86 per cent. The multiple infections were as follows: *Trichuris trichiura* with *Strongyloides stercoralis*, eight times; *Trichuris trichiura* with *Ascaris lumbricoides*, six

times; *Trichuris trichiura* with *Hymenolepis nana*, five times; *Trichuris trichiura* with *Ascaris lumbricoides* and *Strongyloides stercoralis*, four times; *Trichuris trichiura* with *Necator americanus*, three times; *Trichuris trichiura* with *Oxyuris vermicularis*, three times, and one time each of the following: *Ascaris lumbricoides* with *Hymenolepis nana; Oxyuris vermicularis* with *Strongyloides stercoralis; Oxyuris vermicularis* with *Hymenolepis nana; Strongyloides stercoralis* with *Necator americanus; Strongyloides stercoralis* with *Hymenolepis nana; Trichuris trichiura, Ascaris lumbricoides* with *Necator americanus; Trichuris trichiura, Necator americanus* with *Hymenolepis nana; Oxyuris vermicularis, Strongyloides stercoralis* with *Hymenolepis nana; Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis* with *Strongyloides stercoralis; Trichuris trichiura, Ascaris lumbricoides, Necator americanus* with *Hymenolepis nana.*

The youngest individual in this study found to be infected was in this institution. The child was 2 years of age, and had a quadruple infection of *Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis* and *Strongyloides stercoralis.*

The children played with dogs. There was no systematic medical inspection and the stools had never been examined before. Raw washed fruits and vegetables were eaten by the inmates of this institution. Several were dirt eaters, though they were not specified. The previous environments were unknown.

The orphan asylum designated in the tables as No. 3 presented seventy-five individuals for examination, of whom forty-two were males and thirty-three were females, their ages ranging from 4 to 17 years. Of the infected cases, twenty-eight were males and twenty females. The previous environments were not known. The parasitic infections in the forty-eight instances in this group were as follows: *Trichuris trichiura* in thirty-one, or 64.58 per cent., of the cases; *Ascaris lumbricoides* in ten, or 20.83 per cent.; *Oxyuris vermicularis* in eight, or 16.67 per cent.; *Strongyloides stercoralis* in three, or 6.25 per cent., and *Hymenolepis nana* in five, or 10.42 per cent.

The multiple infections showed: *Trichuris trichiura* with *Oxyuris vermicularis* in five instances; *Trichuris trichiura* with *Ascaris lumbricoides* in two instances; *Trichuris trichiura* with *Hymenolepis nana*, and *Ascaris lumbricoides* with *Strongyloides stercoralis* in one instance each.

There were no dogs or animals in this institution. Raw, washed fruits and vegetables were eaten here. There were no crawling children, dirt eaters or finger suckers. The stools were never examined.

The orphan asylum designated in the tables as No. 4 contributed sixty-seven stools for examination from thirty-nine males and twenty-eight females. Their ages ranged from 3 to 14 years. Examination showed twenty-four infections, twelve males and twelve females. The infections were as follows: *Trichuris trichiura* in fifteen, or 62.5 per cent.; *Ascaris lumbricoides* in five, or 20.83 per cent.; *Hymenolepis nana.* in five, or 20.83 per cent., and *Necator americanus* in one, or 4.16 per cent. Double infection occurred twice with the same parasites, namely, *Trichuris trichiura* with *Hymenolepis nana.* The habits and surroundings of the inmates of this institution could not be ascertained, except that raw, washed vegetables were eaten here, and the stools had been examined once for hookworm a number of years ago.

The infant's asylum in this study is designated in the tables as No. 5. There were 110 stools examined from children ranging in age from 3 weeks to 5 years, of whom seventy-three were males and thirty-seven were females. Of those infected, fifteen were males and six were females. No infections occurred in this institution before the second year. The youngest child was 28 months old, and was infected with *Ascaris lumbricoides.* This child suubsequently died of tuberculosis. There were thirteen single infections and eight multiple infections. *Trichuris trichiura* infections were found in nine, or 42.85 per cent.; *Ascaris lumbricoides* in twelve, or 57.14 per cent.; *Oxyuris vermicularis* in three, or 14.28 per cent.; *Hymenolepis nana* in four, or 19.04 per cent., and *Hymenolepis diminuta, Strongyloides stercoralis* and *Necator americanus* in one, or 4.76 per cent., instances each. The multiple infections were: *Trichuris trichiura* with *Strongyloides stercoralis* one time; *Trichuris trichiura* with *Ascaris lumbricoides* two time, and the following combination one time each: *Ascaris lumbricoides* with *Hymenolepis nana; Trichuris trichiura* with *Hymenolepis nana; Trichuris trichiura* with *Ascaris lumbricoides* and *Hymenolepis diminuta;* and *Trichuris trichiura* with *Ascaris lumbricoides* and *Oxyuris vermicularis.* It is of interest that the rat tape worm, *Hymenolepis diminuta,* was found in this institution, and in an individual 3 years old. One of the youngest infections in this series occurred in this institution, namely, the child 28 months old infected with *Ascaris lumbricoides.*

In this institution medical inspection is frequent. The laboratory of the board of health examines stools when a child is suspected, and also makes differential blood examinations in certain conditions when called to do so. There are no dogs or cats in the institution. The children receive both washed and raw vegetables, and fruits and bananas unwashed and raw. Many of these inmates are born in the institution and those entering later come from unsanitary environment. Crawlers, finger suckers and dirt eaters are common here.

The institution designated in the tables as No. 6 is a white waif's home. The stools of fifty-one inmates were examined. All were males whose ages were not obtainable. The percentage of infections in this institution was the greatest in this study, showing forty-six infections in fifty-one individuals, or 90.2 per cent. of the inmates infected. The infections found were: *Trichuris trichiura* in twenty-nine, or 62.83 per cent. of the infections; *Ascaris lumbricoides* in fifteen, or 32.61 per cent.; *Oxyuris vermicularis* in ten, or 21.74 per cent.; *Strongyloides stercoralis* in four, or 8.69 per cent.; *Necator americanus* in four, or 8.69 per cent., and *Hymenolepis nana* in fourteen, or 13.43 per cent., of the instances. The multiple infections were as follows: *Trichuris trichiura* with *Ascaris lumbricoides* in six, or 13.1 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* and *Hymenolepis nana* in six, or 13.1 per cent.; *Trichuris trichiura* with *Hymenolepis nana* in three, or 6.52 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* and *Necator americanus* in two, or 4.36 per cent., and *Strongyloides stercoralis* with *Necator americanus*, *Strongyloides stercoralis* with *Hymenolepis nana*, and *Trichuris trichiura* with *Ascaris lumbricoides*, *Strongyloides stercoralis* and *Hymenolepis nana* in one instance each. Multiple infections occurred in twenty, or 43.48 per cent., of the instances.

The previous environment of the inmates of this institution are rarely sanitary. The population is transient. There is no medical inspection, and while the sanitary conditions are very good, particularly in the pantry and kitchen, the origin, duration of residence and lack of medical inspection may easily be accountable for the heavy infection. There are no dogs or cats in the institution. The vegetables and fruit eaten here are washed.

In the institution designated in the tables as No. 7, the conditions are identical with those in No. 6. In No. 7 the inmates are colored waifs. The stools of seventy-two individuals were examined

showing an infection second in severity only to the white waif's home, as the stools of sixty of the seventy-two inmates were infected, or an 83 per cent. infection. The parasites causing the infection were: *Trichuris trichiura* in forty-two, or 70 per cent.; *Ascaris lumbricoides* in seventeen, or 28.33 per cent.; *Hymenolepis nana* in eleven, or 18.33 per cent.; *Oxyuris vermicularis* in five, or 8.33 per cent.; *Strongyloides stercoralis* in eleven, or 18.33 per cent., and *Necator americanus* in four, or 6.66 per cent. The multiple infections were as follows: *Trichuris trichiura* with *Strongyloides stercoralis* in six, or 10 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* in six, or 10 per cent.; *Trichuris trichiura* with *Hymenolepis nana* in four, or 6.66 per cent.; *Trichuris trichiura* with *Ascaris lumbricoides* and *Strongyloides stercoralis* in two, or 3.33 per cent.; *Trichuris trichiura* with *Oxyuris vermicularis* in two, or 3.33 per cent., and one, or 1.66 per cent., instances of the following combination: *Oxyuris vermicularis* with *Strongyloides stercoralis; Oxyuris vermicularis* with *Hymenolepis nana; Ascaris lumbricoides* with *Strongyloides stercoralis* and *Hymenolepis nana; Ascaris lumbricoides* with *Necator americanus* and *Hymenolepis nana; Ascaris lumbricoides* with *Necator americanus*, and *Necator americanus* with *Hymenolepis nana*. Multiple infections occurred in twenty-six, or 43.33 per cent., of the infections in this institution, the greatest multiple infections occurring in this study.

Age.—The ages of those whose stools were examined ranged from 3 weeks to 18 years, as follows: Under 1 year, fifty-five; 1 year and under 2 years, seventeen; 2 years, three; 3 years, sixteen; 4 years, twenty; 5 years, twenty-one; 6 years, thirteen; 7 years, twenty-four; 8 years, thirty-five; 9 years, thirty; 10 years, thirty-nine; 11 years, forty-eight; 12 years, forty-five; 13 years, forty-two; 14 years, twenty-six; 15 years, twenty-three; 16 years, ten; 17 years, three; 18 years, two. In 123 instances the ages were not given.

There were no infections under 2 years. Two years, two, or 0.33 per cent.; 3 years, nine, or 1.51 per cent.; 4 years, ten, or 1.68 per cent.; 5 years, eight, or 1.34 per cent.; 6 years, ten, or 1.68 per cent.; 7 years, twelve, or 2.01 per cent.; 8 years, twenty-four, 4.03 per cent.; 9 years, seventeen, or 2.85 per cent.; 10 years, seventeen, or 2.85 per cent.; 11 years, twenty-seven, or 4.53 per cent.; 12 years, twenty-six, or 4.36 per cent.; 13 years, twenty-one, or 3.52 per cent.; 14 years,

fifteen, or 2.52 per cent.; 15 years, eight, or 1.34 per cent.; 16 years, three, or 0.41 per cent.; 17 years, one, or 0.16 per cent.; 18 years, one, or 0.16 per cent.

Sex.—Of the 595 stools examined, 365 were from males and 230 from females. The 317 infections included 213, or 67.19 per cent. males and 104, or 32.81 per cent., females; 58.38 per cent. of the males and 45.22 per cent. of the females investigated were infected.

The number of males whose stools were examined at the various ages were: Under 1 year, thirty-five; over 1 year and under 2 years, nine; 2 years, one; 3 years, ten; 4 years, ten; 5 years, fifteen; 6 years, nine; 7 years, fourteen; 8 years, fourteen; 9 years, fourteen 10 years, twenty-two; 11 years, twenty-three; 12 years, twenty; 13 years, eighteen; 14 years, fourteen; 15 years, eight; 16 years, six; 17 years, none; 18 years, none. Of 123 males the ages were not given.

The males infected at the given ages were: 2 years, one, or 0.47 per cent.; 3 years, six, or 2.85 per cent.; 4 years, six, or 2.85 per cent.; 5 years, six, or 2.85 per cent.; 6 years, six, or 2.85 per cent.; 7 years, eight, or 3.8 per cent.; 8 years, nine, or 4.28 per cent.; 9 years, eight, or 3.80 per cent.; 10 years, ten, or 4.74 per cent.; 11 years, fourteen, or 6.7 per cent.; 12 years, fourteen, or 6.7 per cent.; 13 years, five, or 2.37 per cent.; 14 years, seven, or 3.35 per cent.; 15 years, four, or 1.9 per cent.; 16 years, three, or 1.42 per cent. Of 106 infected males the ages were not given.

The number of females whose stools were examined at the various ages were: Under 1 year, twenty; over 1 year and under 2 years, eight; 2 years, two; 3 years, six; 4 years, ten; 5 years, six; 6 years, four; 7 years, ten; 8 years, twenty-one; 9 years, sixteen; 10 years, seventeen; 11 years, twenty-five; 12 years, twenty-five; 13 years, twenty-four; 14 years, twelve; 15 years, fifteen; 16 years, four; 17 years, three; 18 years, two.

The females infected at the various ages were: 2 years, one, or 0.31 per cent.; three years, three, or 0.95 per cent.; 4 years, four, or 1.26 per cent.; 5 years, two, or 0.63 per cent.; 6 years, four, or 1.26 per cent.; 7 years, four, or 1.26 per cent.; 8 years, fifteen, or 4.74 per cent.; 9 years, nine, or 2.84 per cent.; 10 years, seven, or 2.21 per cent.; 11 years, thirteen, or 4.1 per cent.; 12 years, twelve, or 3.76 per cent.; 13 years, sixteen, or 5.05 per cent.; 14 years, eight, or 2.52 per cent.; 15 years, four, or 1.26 per cent.; 16 years, none; 17 years, one, or 0.31 per cent.; 18 years, one, or 0.31 per cent.

TABLE 1.—The Percentages of the Males and Females Examined at the Various Ages

Age, Years	Males			Females		
	Number Examined	Number Infected	Per Cent. Infected	Number Examined	Number Infected	Per Cent. Infected
Under 1	35	0	0	20	0	0
1	9	0	0	8	0	0
2	1	1	100	2	1	50
3	10	6	60	6	3	50
4	10	6	60	10	4	40
5	15	6	40	6	2	33.3
6	9	6	66.6	4	4	100
7	14	8	57	10	4	40
8	14	9	64	21	15	71.5
9	14	8	57	16	9	56.2
10	22	10	45.4	17	7	41.2
11	23	14	61	25	13	52
12	20	14	70	25	12	48
13	18	5	27.8	24	16	66.7
14	14	7	50	12	8	65.7
15	8	4	50	15	4	26.6
16	6	3	50	4	4	100
17	0	0	0	3	2	66.7
18	0	0	0	1	1	100

In 123 instances the ages of males examined were not given.
In 106 instances the ages of males infected were not given.

TABLE 2.—Symptoms Manifested in Infected and Noninfected Cases

Symptom	Number of Cases	Per Cent. of All Cases	Number of Negative Cases	Per Cent. of Negative Cases	Number of Infected Cases	Per Cent. of Infected Cases
Abdominal pain	19	14.3	16	16.8	3	7.9
Constipation	36	27.1	33	34.8	3	7.9
Diarrhea	2	1.5	2	2.12	1	2.63
Alternating diarrhea and constipation	2	1.5	1	1.05		
Capricious appetite	2	1.5	2	2.12		
Good appetite	14	10.52	5	5.26	9	2.37
Poor appetite	7	5.25	6	6.32	1	2.63
Ravenous appetite	52	39.2	42	44.2	10	26.0
Bad breath	43	33.1	35	36.9	8	21.4
Tympanitis	0	0.0	0	0.0	0	0.0
Grinding teeth	21	15.8	14	14.7	7	18 35
Disturbed sleep	14	10.27	10	10.5	4	10.52
Nose picking	20	15.05	14	14.7	6	15.8
Pruritus	1	0.75	1	1.5	0	0.0
Bed wetting	7	5.25	3	3.18	4	10.52
Frequent urination	4	3.0	3	3.18	1	2.63
Convulsions	1	0.75	1	1.05		
Lassitude	49	36.95	38	40.0	11	28.9

Unfortunately, more information could not be secured with regard to the symptoms. In only 133 instances was it possible to secure the symptoms. This included ninety-five negative cases and thirty-eight infected cases.

The comparative frequency of the symptoms attributable to infection with intestinal parasites in the noninfected cases with those infected in this series, even though it is small, should emphasize the necessity of finding either the ova or the parasite itself in making a diagnosis of worms before subjecting an individual, and more especially the infant and small child, to the ordeal of unnecessary medication, as the positive diagnosis is a very simple matter. Especial attention is drawn to the comparative frequency in the noninfected cases with the infected cases where nose picking, grinding of the teeth, disturbed sleep, ravenous appetite and lassitude existed.

THE PARASITES

The parasites found in this series were as follows: *Trichuris trichiura; Ascaris lumbricoides; Oxyuris vermicularis; Strongyloides stercoralis; Necator americanus; Hymenolepis nana* and *Hymenolepis diminuta.* They appeared singly and in various combinations as shown in Table 4.

Trichuris trichiura.—This parasite infected 209 individuals. It was a single offender in 120, or 57.42 per cent., cases; double in sixty-eight, or 32.54 per cent.; triple in eighteen, or 8.61 per cent., and quadruple in three, or 1.43 per cent. Its age incidence was as follows: 2 years, one, or 0.72 per cent.; 3 years, four, or 2.9 per cent.; 4 years, six, or 4.35 per cent.; 5 years, one, or 0.72 per cent.; 6 years, eight, or 5.8 per cent.; 7 years, six, or 4.35 per cent.; 8 years, eighteen, or 13.04 per cent.; 9 years, fifteen, or 10.87 per cent.; 10 years, sixteen, or 11.59 per cent.; 11 years, nineteen, or 13.77 per cent.; 12 years, sixteen, or 11.59 per cent.; 13 years, thirteen, or 9.42 per cent.; 14 years, seven or 5.07 per cent.; 16 years, four, or 2.9 per cent.; 16 years, two, or 1.45 per cent.; 17 years, one, or 0.72 per cent.; 18 years, one, or 0.72 per cent., and in seventy-one instances the ages were not given.

Its period of maximum infection was therefore from 8 to 13 years, inclusive, when over 70 per cent. of its infections occurred.

Males and females were infected at the various ages as follows: 2 years, no males and one female; 3 years, three males and one female; 4 years, five males and one female; 5 years, one male and no female;

6 years, four males and four females; 7 years, five males and one female; 8 years, six males and twelve females; 9 years, seven males and eight females; 10 years, nine males and seven females; 11 years, eleven males and eight females; 12 years, six males and 10 females; 13 years, four males and nine females; 14 years, three males and four females; 15 years, two males and two females; 16 years, one male and one female; 17 years, no males and one female; 18 years, no males and one female.

Ascaris lumbricoides.—This parasite infected eighty-six individuals. It was a single offender in thirty-six, or 41.86 per cent., of its infections; double in twenty-eight, or 32.56 per cent.; triple in nineteen, or 22.09 per cent., and quadruple in three, or 3.49 per cent. Its age incidence was as follows: 2 years, two, or 3.7 per cent.; 3 years, four, or 7.4 per cent.; 4 years, three, or 5.6 per cent.; 5 years, five, or 9.2 per cent.; 6 pears, none; 7 years, five, or 9.2 per cent.; 8 years, seven, or 13 per cent.; 9 years, four, or 7.4 per cent.; 10 years, six, or 11.1 per cent.; 11 years, five, or 9.2 per cent.; 12 years, five, or 9.2 per cent.; 13 years, five, or 9.2 per cent.; 14 years, three, or 5.6 per cent., and in thirty-two instances the ages were not given.

Its period of maximum infection was therefore from 5 to 13 years when over 77 per cent. of its infections occurred.

Males and females were infected at the various ages as follows: 2 years, one male and one female; 3 years, two males and two females; 4 years, two males and one female; 5 years, four males and one female; 7 years, five males and no females; 8 years, three males and four females; 9 years, no males and four females; 10 years, four males and two females; 11 years, three males and 2 females; 12 years, three males and two females; 13 years, two males and three females; 14 years, three males.

Oxyuris vermicularis.—This parasite infected forty-one individuals. It was a single offender in twenty-three, or 56.10 per cent., of the cases; double in fifteen, or 36.58 per cent.; triple in two, or 4.88 per cent., and quadruple in one, or 2.44 per cent. Its age incidence was as follows: 2 years, one, or 3.85 per cent.; 3 years, none; 4 years, three, or 11.54 per cent.; 5 years, one, or 3.85 per cent.; 6 years, three, or 11.54 per cent.; 7 years, two, or 7.69 per cent.; 8 years, four, or 15.38 per cent.; 9 years, one, or 3.85 per cent.; 10 years, none; 11 years, one, or 3.85 per cent.; 12 years, two, or 7.69 per cent.; 13 years, one,

or 3.85 per cent.; 14 years, three, or 11.54 per cent.; 15 years, two, or 7.69 per cent.; 16 years, two, or 7.69 per cent., and in fifteen instances the ages were not given.

There was no period of maximum infection, as the parasite was found rather evenly distributed.

Males and females were infected at the various ages as follows: 2 years, no males and one female; 4 years, two males and one female; 5 years, one male and no female; 6 years, two males and one female; 7 years no males and two females; 8 years, one male, and three females; 9 years, one male and no females; 11 years, no males and one female; 12 years, two males and no females; 13 years, no males and one female; 14 years, two males and one female; 15 years, one male and one female; 16 years, two males and no females.

Strongyloides stercoralis.—This parasite infected forty individuals. It was a single offender in eight, or 20 per cent., of its infections; double in twenty-two, or 55 per cent.; triple in eight, or 20 per cent., and quadruple in two, or 5 per cent. Its age incidence was as follows: 2 years, one, or 4 per cent.; 3 years, none; 4 years, one, or 4 per cent.; 5 years, none; 6 years, one, or 4 per cent.; 7 years, three, or 12 per cent.; 8 years, one, or 4 per cent.; 9 years, three, or 12 per cent.; 10 years, four, or 16 per cent.; 11 years, two, or 8 per cent.; 12 years, three, or 12 per cent.; 13 years, three, or 12 per cent.; 14 years, one, or 4 per cent.; 15 years, two, or 8 per cent., and in fifteen instances the ages were not given.

Its period of maximum infection was therefore from 7 to 13 years, inclusive, when 76 per cent. of the infections occurred.

Males and females were infected at the various ages as follows: 2 years, no males and one female; 4 years, no males and one female; 6 years, one male and no females; 7 years, one male and two females; 8 years, one male and no females; 9 years, one male and two females; 10 years, two males and two females; 11 years, two males and no females; 12 years, three males and no females; 13 years, no males and three females; 14 years, no males and one female; 15 years, no males and two females.

Necator americanus.—This parasite infected eighteen individuals. It was a single offender in 5, or 27.78 per cent., of its infections; double in seven, or 38.89 per cent.; triple in five, or 27.78 per cent., and quadruple in one, or 5.55 per cent. Its age incidence was as follows: 2 years, none; 3 years, one, or 10 per cent.; 7 years, one, or

TABLE 3.—Results of One Examination of Feces for Animal Parasites

Institution	Number Examined	Number of Infections		Single Infection		Double Infection		Triple Infection		Quadruple Infection		Trichuris Trichiura		Ascaris Lumbricoides		Oxyuris Vermicularis		Strongyloides Stercoralis		Necator Americanus		Hymenolepis Nana		Hymenolepis Diminuta	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
No. 1	95	17	17.89	15	15.78	2	2.1	0	0 0	0	0.0	10	10.0	1	1.05	6	6.31	0	0.0	0	0.0	2	2.1	0	0.0
No. 2	125	101	80.08	62	49.60	30	24.0	7	5.68	2	1.6	73	58.40	26	20.80	9	7.20	21	16.80	8	6.40	14	11.20	0	0.0
No. 3	75	48	64.0	39	52.0	9	12.0	0	0.0	0	0.0	31	41.33	10	13.33	8	10.66	3	4.0	0	0.0	5	6.66	0	0 0
No. 4	67	24	35.82	22	32.83	2	2.98	0	0.0	0	0.0	15	22.37	5	7.46	0	0.0	0	0.0	1	1.49	5	7.46	0	0.0
No. 5	110	21	19.09	13	11.81	6	5.45	2	1.81	0	0.0	9	8.18	12	10.9	3	2.72	1	0.9	1	0.9	4	3.63	1	0.9
No. 6	51	46	90.2	26	50.98	11	21.58	8	15.88	1	1.96	29	56.86	15	29.41	10	19.60	4	7.84	4	7.84	14	27.45	0	0.0
No. 7	72	60	83.33	34	47.22	22	30.55	4	5.55	0	0.0	42	58.34	17	23.61	5	6.94	11	15.27	4	5.55	11	15.27	0	0 0
Total	595	317	53.27	211	35.47	82	13.78	21	3.53	3	0.5	209	35 12	86	14.62	41	6.89	40	6.72	18	3.0	55	9.25	1	0.17

TABLE 4.—Results of Second Examination of Feces for Animal Parasites

Institution	Number Infected	Single Infection		Double Infection		Triple Infection		Quadruple Infection		Trichuris Trichiura		Ascaris Lumbricoides		Oxyuris Vermicularis		Strongyloides Stercoralis		Necator Americanus		Hymenolepis Nana		Hymenolepis Diminuta	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
No. 1	17	15	88.25	2	11.75	0	0.0	0	0.0	10	58.82	1	5.88	6	35.29	0	0.0	0	0.0	2	11.76	0	0.0
No. 2	101	62	61.39	30	29.7	7	6.93	2	1.98	73	72.29	26	25.34	9	8.91	21	20.89	8	7.92	14	13.86	0	0.0
No. 3	48	39	81.25	9	18.75	0	0.0	0	0.0	31	64.58	10	20.83	8	16.67	3	6.25	0	0.0	5	10.42	0	0.0
No. 4	24	22	91.66	2	8.33	0	0.0	0	0.0	15	62.5	5	20.83	0	0.0	0	0 0	1	4.16	5	20.83	0	0.0
No. 5	21	13	61.90	6	28.57	2	9.52	0	0.0	9	42.85	12	57.14	3	14.28	1	4.76	1	4.76	4	19.04	1	4.76
No. 6	46	26	56.52	11	23.91	8	17.39	1	2.17	29	62.83	15	32.61	10	21.74	4	8.69	4	8.69	14	13.43	0	0.0
No. 7	60	34	56.66	22	36.66	4	6.66	0	0.0	42	70.0	17	28.33	5	8.33	11	18.33	4	6.66	11	18.33	0	0.0
Total	317	211	66.56	82	25.87	21	6.62	3	0.95	209	65 9	86	27.13	41	12.93	40	12.62	18	5.68	55	17.35	1	0.32

10 per cent.; 9 years, one, or 10 per cent.; 11 years, one, or 10 per cent. 12 years, one, or 10 per cent., 13 years, three, or 30 per cent.; 15 years, two, or 20 per cent., and in eight instances the ages were not given.

No period could be considered as of maximum infection with this parasite.

Males and females were infected at the various ages as follows: 3 years, no males and one female; 7 years, one male and no females; 9 years, one male and no females; 11 years, no males and one female; 12 years, no males and one female; 13 years, one male and two females; 15 years, no males and two females.

Hymenolepis nana.—This parasite infected fifty-five individuals. It was a single offender in nineteen, or 34.55 per cent., of its infections; double in twenty-four, or 43.64 per cent.; triple in ten, or 18.18 per cent., and quadruple in two, or 3.63 per cent. Its age incidence was as follows: 3 years, three, or 10 per cent.; 4 years, one, or 3.33 per cent.; 5 years, three, or 10 per cent.; 6 years, one, or 3.33 per cent.; 7 years, one, or 3.33 per cent.; 8 years, two, or 6.67 per cent.; 9 years, one, or 3.33 per cent.; 11 years, five, or 16.67 per cent.; 12 years, five, or 16.67 per cent.; 13 years, two, or 6.67 per cent.; 14 years, five, or 16.67 per cent., and 16 years, one, or 3.33 per cent. In twenty-five instances the ages were not given.

In the infection of this parasite there also seemed no period of maximum infection, though 70 per cent. of the infections occurred above the age of 8 years.

Males and females were infected at the various ages as follows: 3 years, three males and no females; 4 years, one female; 5 years, two males and one female; 6 years, one male and no females; 7 years, one male and no females; 8 years, one male and one female; 9 years, no males and one female; 11 years, three males and two females; 12 years, four males and one female; 13 years, no males and two females; 14 years, one male and four females; 16 years, one male and no females.

Hymenolepis diminuta.—This parasite was found in one instance in which there was a triple infection with *Trichuris trichiura* and *Ascaris lumbricoides*. The individual infected was a female, 3 years of age.

BLOODS

The bloods of 307 individuals included in this study were examined. Differential counts were made from the bloods of 124 of the negative cases and 183 of the positive cases. Table 5 gives the num-

TABLE 5.—Number of Infected and Noninfected Males and Females of Various Ages

Age	Institution 1		2		3		4		5		Total	
	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected	Infected	Not Infected
Under 1 year												
Male	..	..	..	..	..	..	..	..	..	35	..	35
Female	..	..	..	..	..	..	..	..	..	20	..	20
1 year												
Male	..	..	..	..	..	..	..	..	..	9	..	9
Female	..	1	..	..	..	..	..	..	..	7	..	8
2 years												
Male	..	..	..	..	..	..	..	..	1	..	1	.
Female	..	..	1	..	..	..	..	.	..	1	1	1
3 years												
Male	..	..	..	..	..	..	1	..	5	4	6	4
Female	..	1	..	1	..	..	..	..	3	1	3	3
4 years												
Male	..	..	1	..	..	2	..	..	5	2	6	4
Female	..	1	1	1	1	1	..	1	2	2	4	6
5 years												
Male	..	1	..	..	2	..	..	1	4	7	6	9
Female	1	..	..	2	..	..	..	1	1	1	2	4
6 years												
Male	1	2	3	..	2	..	..	1	..	..	6	3
Female	..	..	1	..	3	..	..	..	..	..	4	..
7 years												
Male	..	3	5	..	1	..	2	3	..	..	8	6
Female	..	1	2	..	1	1	1	4	..	..	4	6
8 years												
Male	..	1	3	..	5	1	1	3	..	..	9	5
Female	1	3	9	1	4	2	1	0	..	..	15	6
9 years												
Male	..	1	7	..	..	1	1	4	..	..	8	6
Female	..	4	6	1	1	1	2	1	..	..	9	7
10 years												
Male	1	5	4	..	4	2	1	5	..	..	10	12
Female	..	4	6	2	..	2	1	2	..	..	7	10
11 years												
Male	2	4	3	..	6	1	3	4	..	..	14	9
Female	2	3	8	3	3	2	..	4	..	..	13	12
12 years												
Male	2	3	6	..	4	2	2	1	..	..	14	6
Female	1	5	6	4	2	2	3	2	..	..	12	13
13 years												
Male	..	5	3	..	1	3	1	5	..	..	5	13
Female	1	5	9	3	3	..	3	..	..	..	16	8
14 years												
Male	2	5	3	..	2	2	..	..	..	..	7	7
Female	.	3	7	..	..	..	1	1	..	..	8	4
15 years												
Male	..	4	3	..	1	..	..	.	.	..	4	4
Female	1	7	2	3	1	1	..	..	..	..	4	11
16 years												
Male	1	3	2	..	..	..	..	..	..	..	3	3
Female	..	2	..	1	1	1	..	..	..	..	..	4
17 years												
Male	..	..	..	..	..	..	..	..	..	..	..	..
Female	..	..	..	2	1	..	..	..	..	..	1	2
18 years												
Male	..	..	..	..	..	..	..	..	..	..	..	..
Female	1	1	..	..	..	..	..	..	..	..	1	1
Totals	17	78	101	24	48	27	24	43	21	89	211	261

bers and averages of bloods for the different ages for both positive and negative cases, and the average of all the bloods of the negative cases as well as the average of all the bloods of the positive cases. A comparison of the average differential count for all ages shows an increase in the eosinophils of the infected cases of 3.5 per cent. over the average of the noninfected cases, the increase apparently being at the expense of the neutrophils.

In the study of the eosinophils for the averages at the different ages, the variations in negative cases were from 0 to 4.8 per cent. In only three instances, namely, for the ages of 4, 8 and 11 years, respectively, were the averages above 4 per cent., and in each of these instances the average was heightened by one or two cases with marked eosinophilia. On the other hand, the averages for the different ages in the positive cases, with the exception of the eighteenth year, where only one case was examined, and where the eosinophils were 2 per cent., ranged from 4.7 to 9.2 per cent., clearly showing the value of the eosinophilia as an aid in the diagnosis of intestinal parasitic infections.

Further interest is evidenced in the study of the eosinophils of the individual cases. Of the infected cases in three instances there was a 28 per cent. eosinophilia; in one instance there was a 23 per cent. eosinophilia; in one a 22 per cent., in two an 18 per cent., in one a 17 per cent., in one a 16 per cent., in one a 15 per cent., in eight a 14 per cent., in three a 13 per cent., in seven a 12 per cent., in five an 11 per cent., in seven a 10 per cent., in eleven a 9 per cent., in eight an 8 per cent., in twelve a 7 per cent., in fifteen a 6 per cent., in nine a 5 per cent. and in fourteen a 4 per cent. eosinophilia; or in 59.5 per cent. the percentage of eosinophils was four or more; in 51.9 per cent. it was five or more; in 47 per cent., six or more; in 38.7 per cent., seven or more; in 32.2 per cent., eight or more; in 27.8 per cent., nine or more; in 21.8 per cent., 10 or more; in 18 per cent., 11 or more; in 15.3 per cent., 12 or more; in 11.4 per cent., 13 or more; in 9.8 per cent., 14 or more; in 5.4 per cent., 15 or more; in 4.9 per cent., 16 or more; in 4.3 per cent., 17 or more; in 3.8 per cent., 18 or more; in 2.7 per cent., 22 or more; in 2.1 per cent., 23 or more, and in 1.6 per cent. of the infected cases there was a 28 per cent. eosinophil count.

In the noninfected cases in one instance there was an 18 per cent. eosinophilia; in one a 16 per cent. eosinophilia; in two a 13 per cent.; in one a 12 per cent.; in one a 10 per cent.; in four an 8 per cent.; in

four a 7 per cent.; in four a 6 per cent.; in five a 5 per cent., and in seventeen a 4 per cent. eosinophilic count; or, in 32 per cent. of the cases the eosinophils numbered 4 per cent. or more; in 18.5 per cent., 5 or more; in 14.5 per cent., 6 or more; in 11.2 per cent., 7 or more; in 8 per cent., 8 or more; in 4.8 per cent., 10 or more; in 4 per cent., 12 or more; in 3.2 per cent., 13 or more; in 1.6 per cent., 16 or more, and in 0.8 per cent. of the noninfected cases there was an 18 per cent. eosinophil count. These results are of especial interest, because, while an eosinophilia should suggest an intestinal parasitic infection, it is not pathognomonic of it.

It is of interest to note that of the positive cases all the eosinophilias from 15 per cent. and above were in the same institution (No. 2).

In studying the eosinophils in their relation to the infections of the various parasites singly and in combination, the following was revealed: The maximum eosinophilia in the single infection by the *Trichuris trichiura* was 15 per cent., which occurred in one instance. While, on the other hand, there were two infections by this parasite in which there were no eosinophils shown in the count. The eosinophil count was 4 per cent. or less, as follows: In fourteen instances the percentage was 1 per cent.; in nine it was 2 per cent.; in six it was 3 per cent., and in seven it was 4 per cent.

The maximum eosinophilia in connection with the single infection with the *Ascaris lumbricoides* was 16 per cent.; the minimum 0.0 per cent. The eosinophil count was 4 per cent. or less, as follows: One patient had a 1 per cent., four patients had a 4 per cent., two patients had a 3 per cent., and three patients had a 4 per cent.

The *Oxyuris vermicularis* showed a maximum of 11 per cent. eosinophils in single infections and a minimum of 0.0 per cent. The eosinophil count was 4 per cent. or less, as follows: In four instances there was one case with 1 per cent.; three cases with 2 per cent.; two cases with 3 per cent., and two cases with 4 per cent.

The *Strongyloides stercoralis* in single infection showed a 14 per cent. maximum eosinophil count and a 0.0 per cent. minimum count. The eosinophil count was 4 per cent. or less, as follows: There was one case with 1 per cent., and one case with 2 per cent.

The *Necator americanus* occurred only in two instances in single infection, once with a 12 per cent. and the other time with a 13 per cent. eosinophil count. It is of interest to note that the lowest eosinophil counts with this parasite in combination under 4 per cent. were

1 per cent. with a mixed infection with *Trichuris trichiura* and *Hymenolepis nana* in one instance, and in a double infection with *Strongyloides stercoralis* in which the eosinophil count was 3 per cent.

TABLE 6.—Parasites and Number of Times Each Was Found Alone or Associated with Others

Infections	Institutions							Total
	1	2	3	4	5	6	7	
Trichuris trichiura	8	40	23	13	3	11	22	120
Ascaris lumbricoides	0	12	7	5	6	0	6	36
Oxyuris vermicularis	6	2	3	0	1	10	1	23
Strongyloides stercoralis	0	6	2	0	0	1	1	8
Necator americanus	0	1	0	1	1	1	1	5
Hymenolepis nana	1	3	4	3	2	3	3	19
Hymenolepis diminuta	0	0	0	0	0	0	0	0
Trichuris trichiura, Ascaris lumbricoides	1	6	2	0	2	6	6	23
Trichuris trichiura, Oxyuris vermicularis	0	3	5	0	0	0	2	10
Trichuris trichiura, Strongyloides stercoralis	0	8	0	0	1	0	6	15
Trichuris trichiura, Necator americanus	0	3	0	0	0	0	0	3
Trichuris trichiura, Hymenolepis nana	1	5	1	2	1	3	4	17
Ascaris lumbricoides, Oxyuris vermicularis	0	0	0	0	1	0	0	1
Ascaris lumbricoides, Strongyloides stercoralis	0	0	1	0	0	0	0	1
Ascaris lumbricoides, Necator americanus	0	0	0	0	0	0	1	1
Ascaris lumbricoides, Hymenolepis nana	0	1	0	0	1	0	0	2
Oxyuris vermicularis, Strongyloides stercoralis	0	1	0	0	0	0	1	2
Oxyuris vermicularis, Hymenolepis nana	0	1	0	0	0	0	1	2
Strongyloides stercoralis, Necator americanus	0	1	0	0	0	1	0	2
Strongyloides stercoralis, Hymenolepis nana	0	1	0	0	0	1	0	2
Necator americanus, Hymenolepis nana	0	0	0	0	0	0	1	1
Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis	0	0	0	0	1	0	0	1
Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis	0	4	0	0	0	0	2	6
Trichuric trichiura, Ascaris lumbricoides, Necator americanus	0	1	0	0	0	2	0	3
Trichuris trichiura, Ascaris lumbricoides, Hymenolepis nana	0	0	0	0	0	6	0	6
Trichuris trichiura, Ascaris lumbricoides, Hymenolepis diminuta	0	0	0	0	1	0	0	1
Trichuris trichiura, Necator americanus, Hymenolepis nana	0	1	0	0	0	0	0	1
Ascaris lumbricoides, Strongyloides stercoralis, Hymenolepis nana	0	0	0	0	0	0	1	1
Ascaris lumbricoides, Necator americanus, Hymenolepis nana	0	0	0	0	0	0	1	1
Oxyuris vermicularis, Strongyloides stercoralis, Hymenolepis nana	0	1	0	0	0	0	0	1
Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis, Strongyloides stercoralis	0	1	0	0	0	0	0	1
Trichuris trichiura, Ascaris lumbricoides, Strongyloides stercoralis, Hymenolepis nana	0	0	0	0	0	1	0	1
Trichuris trichiura, Ascaris lumbricoides, Necator americanus, Hymenolepis nana	0	1	0	0	0	0	0	1

Hymenolepis nana showed in a single infection a maximum of 18 per cent. eosinophils, and in three instances the percentage was zero. The eosinophil count was 4 per cent. or less, as follows: There were three instances of 1 per cent., and one of 2 per cent.

In only one case was the *Hymenolepis diminuta* found, in which instance it was in a triple infection with *Trichuris trichiura* and *Ascaris lumbricoides*. The eosinophil count in this case was 13 per cent.

The maximum eosinopil count in the double infection was 28 per cent. in two instances, both being combined infections with the *Trichuris trichiura* and *Strongyloides stercoralis.* The minimum eosinophilia in this combination was 1 per cent. in one instance. There were no others below 6 per cent.

The minimum eosinophil count in a double infection was zero in a mixed infection of *Trichuris trichiura* with *Ascaris lumbricoides.* In two double infections the count was 1 per cent., namely, *Trichuris trichiura* with *Ascaris lumbricoides,* and *Trichuris trichiura* with *Oxyuris vermicularis.* In four instances there was a 2 per cent.; in two instances a 3 per cent., and in three instances a 4 per cent. eosinophil count.

TABLE 7.—Results of Examination of Blood Infected and Noninfected Cases

Age	Not Infected						Infected					
	No. Cases	Neutrophils, per Cent.	Small Mononuclears, per Cent.	Large Mononuclears, per Cent.	Eosinophils, per Cent.	Basophils, per Cent.	No. Cases	Neutrophils, per Cent.	Small Mononuclears, per Cent.	Large Mononuclears, per Cent.	Eosinophils, per Cent.	Basophils, per Cent.
1 yr.	1	44.0	56.0	—	—	—	0	—	—	—	—	—
2 yrs.	0	....	....	—	—	—	1	40.0	45.0	5.0	9.0	1
3 yrs.	2	56.5	42.5	0.5	0.5	2	7	47.6	39.9	6.0	6 3	2
4 yrs.	5	55.2	38.8	0.1	5.0	0	10	50.5	39.3	5.5	4.7	0
5 yrs.	3	52.7	42.3	0.2	3.0	0	8	53.4	36.4	4.1	5.7	4
6 yrs.	2	59.0	35.0	2.5	3.5	0	10	58.2	32.1	2.1	7.0	6
7 yrs.	5	62.8	32.4	1.8	2.8	2	9	57.7	31.0	1.3	9.3	2
8 yrs.	8	57.1	35.6	2.3	4.8	2	22	57.3	35.4	9.0	6.3	1
9 yrs.	8	57.9	35.6	3.3	3.1	1	13	57.0	33.2	1.2	8.4	2
10 yrs.	13	57.6	37.6	1.8	3.0	0	15	63.0	30.3	1.1	5.5	1
11 yrs.	13	60.6	32.6	2.5	4.2	1	23	57.8	35.3	2.1	4.7	1
12 yrs.	15	63.4	32 1	2.1	2.3	1	21	57.2	23.7	2.2	6.5	4
13 yrs.	16	61.1	36.0	1.4	1.4	1	16	54.9	36.4	1.0	7.6	1
14 yrs.	10	58.4	37.3	1.5	2.8	0	14	58.3	35.8	1.0	4.8	1
15 yrs.	15	63.1	33.5	1.4	1.9	1	7	55.6	38.4	6.0	5 3	1
16 yrs.	7	60.8	35.4	2.4	1.4	0	3	57.4	32.6	3.0	7.0	0
17 yrs.	0	....	....	....	....	..	3	62.0	28.0	2.0	8.0	0
18 yrs.	1	66.0	32.0	0.2	0.0	0	1	72.0	25.0	1.0	2.0	0
Aver. for all cases	124	59.92	35.39	1.9	2.71	0.08	183	56.27	35.26	2.01	6.27	0.19

In the triple infections the maximum eosinophil count was 28 per cent. in a combined infection of *Trichuris trichiura, Ascaris lumbricoides* and *Strongyloides stercoralis.* The minimum count was zero with an infection of *Trichuris trichiura, Ascaris lumbricoides* and *Oxyuris vermicularis;* and 1 per cent. in a triple infection of *Trichuris trichiura, Necator americanus* and *hymenolepis nana.*

The blood was examined in two quadruple infections, the eosinophil counts being 9 and 8 per cent., respectively. In the former the combined infections were of the *Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis* and *Strongyloides stercoralis,* and in the latter *Trichuris trichiura, Ascaris lumbricoides, Necator americanus* and *Hymenolepis nana.* It is of especial interest that in the former infection the individual was a child, 2 years of age.

CONCLUSIONS

1. In this study the frequency of the infections by the various parasites were: *Trichuris trichiura, Ascaris lumbricoides, Hymenolepis nana, Oxyuris vermicularis, Strongyloides stercoralis, Necator americanus* and *Hymenolepis diminuta.*

2. The hygiene, general care, duration of residence in the institution, previous environment, association with dogs, eating of dirt, absence of systematic medical inspection, seemed to have a direct bearing on the frequency of intestinal parasitic infection.

3. The two institutions where systematic medical inspections were made even though there were many crawlers in one of these institutions, showed the lowest percentages of infections, namely, 17.89 per cent. and 19.00 per cent., respectively.

4. The two institutions with the heaviest infections were those where the population was transient and where there were no medical inspections, the infections being 90.2 and 83.33 per cent., respectively.

5. The youngest individual infected was in the least sanitary of the institutions investigated, this child was 2 years of age and had a quadruple infection of *Trichuris trichiura, Ascaris lumbricoides, Oxyuris vermicularis,* and *Strongyloides stercoralis.*

6. The relation of age to infection seemed negligible except that there were no infections among the infants; in other words, before the period of exposure there were no infections. The maximum number of infections occurred at the age of 11 years.

7. Males were infected over 13 per cent. more frequently than were females. Again the element of exposure seems to have had some influence. The maximum ages of the infections in boys were 11 and 12 years, and for girls, 13 years. Infections, however, were rather generally distributed over several years.

8. No information of value was obtained in studying the incidence at the different ages, except that the greatest incidence in boys was fourteen infections in twenty boys, or 70 per cent. of infections at the age of 12 years, and for the girls fifteen in twenty-one, or 71.5 per cent. of infections at the age of 8 years.

9. The symptoms usually attributed to "having worms" were as frequent, and some a little more frequent, in those not infected than in those infected.

10. One infection with the rat tape worm (*Hymenolepis diminuta*) was found in a female child, 3 years of age. The infection occurred in a triple infection of *Hymenolepis diminuta* and *Trichuris trichiura* with *Ascaris lumbricoides*.

11. An eosinophilia is a valuable aid in the diagnosis of infection with intestinal parasites. It is, however, not pathognomonic of, and its absence does not exclude, an infection.

12. The eosinophils were found as high as 28 per cent. in the positive cases, and 18 per cent. in the negative cases. They were 4 per cent. or above 59.5 per cent. of the positive cases, and 4 per cent. above in 32 per cent. of the negative cases. It was of interest that all the eosinophilias of 15 per cent. and above in the positive cases occurred in the same institution.

13. No parasite in this study seemed to have a definite eosinophilia.

14. It was shown that the infection by any of the intestinal parasites in this study could exist with an absence of eosinophils. This was found in their single infections with the exception of *Necator americanus* and *Hymenolepis diminuta*. *Necator americanus* was found in single infections in only two cases, in which instances the eosinophils were 12 and 13 per cent., respectively. However, in mixed infection of this parasite with *Trichuris trichiura* and *Hymenolepis nana* the eosinophils were as low as 1 per cent.

The other exception was the *Hymenolepis diminuta* which was not found in single infection, but in combination with *Trichuris trichiura* and *Ascaris lumbricoides* in which case the eosinophils numbered 13 per cent.

15. Minimum eosinophil counts in double and triple infections were zero, while the maximum was 28 per cent. in each.

16. The two quadruple infections in which blood examinations were done showed an 8 and 9 per cent. eosinophilia, respectively.

17. The diagnosis of intestinal parasitic infection is an extremely easy matter. The symptoms usually ascribed to worms are as frequent in those not infected as in those infected. Blood examinations showing eosinophilias are valuable diagnostic aids, but the only way to make a positive diagnosis is by either finding the parasite or the ova in the stool of the individual. Vermifuges should, therefore, not be given to the defenseless child unless it is known that an infection exists and its character is ascertained when the specific drug may be given to combat the infection.

DISCUSSION

Dr. Sedgwick: I think it is very necessary to drive home the point that Dr. DeBuys has brought out, that institutions should have thorough medical inspection. Many institutions are carried on without physical examinations of any real value. In many institutions the possibility of the existence of worms is entirely overlooked. Physicians are likely not to think them as important as the laity does. Up in Minnesota perhaps we do not have as many parasites as are found in the South, but I think that if we looked for them systematically we would find more. This subject has been well presented.

Dr. Brennemann: I think it is rather interesting that one of the men who has done most of the work on intestinal parasites is a biologist. I sometimes wonder if the subject is not more interesting from a biological than from a medical standpoint. I would like to ask Dr. DeBuys whether after his experience he makes it a practice to examine many stools in private practice, and I would like to know whether he has found symptoms which point to the probable presence of worms. I think most of us in the United States when we think of worms think of the *Ascaris lumbricoides*. Nevertheless Dr. Kerley says in his book that he has seen only four or five instances in which he found the *Ascaris lumbricoides* in New York. That is my own experience. The children coming under my observation who have harbored these parasites have not shown any characteristic symptoms until a parasite was demonstrated and then after the administration of santonin more of the parasites would be found. I would like to ask Dr. DeBuys whether he has noted any characteristic symptoms and whether he thinks it is worth while looking for intestinal parasites as a routine procedure.

Dr. Schloss: The occurrence of symptoms due to intestinal parasites is variable and the symptoms seem to depend to some degree on the grade of infection. A few years ago, I did considerable work on the occurrence of intestinal parasites in New York, with special reference to the symptomatology. In my series of cases infection with *Trichuris trichiura* was very common, but these parasites are very rarely responsible for symptoms. A few cases are in the literature where they apparently caused diarrhea with a great deal of mucus and blood in the stools. I have seen one such case. These parasites are, however, very difficult to eliminate.

Oxyuris vermicularis is also a very common parasite and the diagnosis is often a matter of some difficulty. The ova are very rarely found in the stools, more often the adult parasite. In a suspicious case the only satisfactory method of diagnosis is to give a dose of santonin and to examine the stools very carefully for the parasites themselves. *Oxyuris* very rarely causes any symptoms

beyond rectal itching. In some cases, one finds the symptoms to which Still called attention some time ago, namely, a distinct pain in the right iliac fossa. This has been a matter of considerable discussion and some writers believe that the presence of these parasites in the appendix and cecum may be definitely responsible for appendicitis.

In my series of cases the dwarf tapeworm was very common and there were several instances in which it was apparently responsible for very marked anemia. In general, however, the cases in which symptoms occurred were relatively few in proportion to the number infected.

It is an interesting fact that despite the frequency of intestinal parasites in dispensary patients, they are very uncommon in private practice. I have seen but three or four cases.

Dr. Carpenter: Dr. DeBuys' paper is one of much importance, because this subject is so frequently overlooked. In a large orphan asylum in Philadelphia, there is at present twenty children whose stools show double infection with the ova of the *Hymenolepsis nana*, and the *Trichuris trichiura*. These children have undoubtedly had this infection for many months. In spite of a repeated search the worm itself has not been found, but ova can be found without difficulty. Many forms of treatment have been instituted, but ova are still present. I would like to know what Dr. DeBuys' experience has been in freeing the children from *hymenolepsis nana*, and what treatment he has found most successful? Particular attention has been paid to see if the health of these children was impaired, but the blood counts in all the infected children was practically normal. This is of interest, as it is so frequently stated this tapeworm produces severe symptoms. A careful study was made to determine how the infection was acquired. The rats in the institution which were trapped were negative as far as these parasites were concerned. The children infested were of the age commonly known as creepers, and the tapeworms most likely acquired by hand-to-mouth infection from crawling on the floor.

Dr. Marriott: The point has been brought out that the *Oxyuris* may cause symptoms referred to the right iliac region. G. F. Still has drawn attention to the fact the parasites may multiply in the appendix. We recently observed a boy of 8 years in a typical attack of appendicitis. The appendix was removed and found to be a veritable nest of *Oxyuris*. There was little or no inflammatory process. The symptoms disappeared after the appendectomy. The usual methods of treatment for the parasites would, of course, have been unavailing in such an instance.

Dr. Mitchell: I would like to speak on the question as to whether intestinal parasites cause symptoms. Formerly I had been accustomed to believe that they caused little if any trouble. Recently I had the opportunity to observe a large number of children in France, where the percentage of infestion with *Ascaris lumbricoides* was high. Here my ideas changed somewhat. Many of these children had intestinal symptoms which cleared up when the infestion was overcome. Certainly anorexia and nausea, followed and relieved by the vomiting of a worm are to be classed as symptoms. It seemed also that the children who had large numbers of these parasites showed an improvement in general nutrition following treatment.

Dr. Park: We saw a large number of cases of intestinal parasites in France. We have never seen anything to compare with them in point of numbers in this country. Dr. Crawford and I made an attempt to find out whether infestation with round worm produced symptoms or not. We examined

the stools of all children in whose cases infection with round worms was suspected. When we discovered that it was present, we proceeded as follows. First we made careful notes in regard to a list of symptoms commonly ascribed to infection with round worms in order to establish an accurate record of the symptoms before any treatment was established. We then put the child on a good diet and regimen and endeavored to make the conditions under which the child lived as nearly normal as possible. At the end of three weeks we again made careful notes in regard to the list of symptoms previously inquired into in order to determine which of them had disappeared and which persisted; in other words, to find out which symptoms of the list were to be referred to the round worm infection and which to other conditions. Finally we put the child on treatment for the round worm infestation in the expectation of inquiring into the list of symptoms three weeks after the round worms had been removed. The symptoms, which were still present after the diet and regimen had been corrected and which disappeared with the removal of the round worm, might well be attributed to the latter. We were almost never able to rid the child of the worms, however, in all probability for the reason that the preparations of santonin available in France were not effective. Nevertheless, we were able to obtain evidence that very few if any symptoms generally ascribed to ascaris infection could be actually referred to that cause. For in almost every one of the fifty cases studied all the symptoms disappeared after correction of the diet and the regimen. Moreover, we saw a number of children, some of them very young, whose stools were filled with ascaris eggs and who frequently passed large numbers of ascarides without any evidence, objective or subjective, of disturbance in the general health.

Dr. Stiles once told me that he had seen cases of ascaris infection in the mountainous districts of the South which showed the most severe cachexia and that ascaris infection undoubtedly could be the cause of most grave symptoms. Undoubtedly infestation with ascaris causes the most severe symptoms in puppies and often is responsible for their death. I have had considerable experience with ascaris infestation in puppies, characterized by progressive emaciation, convulsions and finally death. At necropsy the stomach and upper part of the small intestine were found filled with round worms.

In France we had under observation a child less than 1 year old who passed 132 round worms in three days and another child, only 3 months old, who vomited a large round worm. The only symptoms which we thought might be ascribed to infection with round worms were indefinite abdominal pains and vomiting, usually of the round worms themselves.

Dr. DeBuys: I did not attempt to go into the complete analysis of intestinal parasites, but only as they were related to this investigation. Nor was any mention made of treatment as it was not included in the study. I did want to emphasize the fact that a diagnosis of intestinal parasites could not be made on symptoms usually attributed to worms. However, I did not want to give the impression that infestation by intestinal parasites did not cause symptoms. The American hookworm, for instance, causes severe anemia and interferes with growth and development both physical and mental. I have seen *Ascaris lumbricoides* cause grave symptoms. One should remember the predilection of the *Ascaris lumbricoides* to enter small openings. I have seen it and know of four instances in which it was found in the appendix at operation. At the operation, the appendix was seen moving. I have known the *Ascaris lumbricoides* to cause intestinal obstruction, and in one instance where an end to end anastomosis was done because of gangrene of the bowel and a Murphy button used after

resection, the worms later used the Murphy button as a nucleus and caused another obstruction with a fatal termination. Necropsy showed the condition existing as described.

We have seen many cases of malnutrition in which the infestation was rather great, and we have found many children under these conditions to be under size. Pruritus was found in only one instance in this series which was a case of infestation. In some of the infestations, the stools were found to contain microscopic blood. This was more likely to be the case in the infestation with the *Strongyloides stercoralis* and the *Necatar americanus*. In examining the stools for *Oxyuris vermicularis* better results are obtained if a formed stool is secured and its surface washed, the washings being examined. The reasons for this is the habitat of this worm.

It is a routine with me to examine the stools for intestinal parasitic infections of all patients coming from Central or South America.

The dwarf tapeworm or *Hymenolepis nana* may be carried about by rats. We found a large percentage of the cases infected with the dwarf tapeworm in the three institutions that were most unsanitary. It is advisable not to allow two persons to sleep in the same bed if one is infested with the dwarf tapeworm as the infection is very easily carried. In regard to the treatment of the infection with the *Hymenolepis nana*, male fern is the drug most frequently used.

Regarding the finding of the *Oxyuris* in the appendix, it is of interest inasmuch as this site is rather high up for this parasite to be found.

DEXTROSE TOLERANCE IN ATROPHIC INFANTS

H. F. HELMHOLZ AND L. W. SAUER
CHICAGO

As a rule the tolerance of individuals to sugar was determined either by testing how much could be given by mouth or how much could be injected at one time subcutaneously or intravenously before sugar appeared in the urine. It was recognized that these methods are open to objections. In order to overcome these objections Woodyatt[1] constructed an apparatus permitting of a continuous intravenous injection of solutions at any desired rate of speed. This apparatus consists of a syringe automatically filling and discharging. In this way he was able to express the tolerance of individuals to dextrose much more accurately than had been possible before. He and his co-workers[2] found that the appearance of sugar in the urine depends on the amount of sugar injected in a unit of time, independent of the concentration of the sugar solution. The tolerance is expressed in grams glucose which just can be injected per 1 kg. body weight in one hour at a uniform rate of flow, without leading to the excretion of sugar in the urine. For normal adults, dogs and rabbits this tolerance is from 0.8 to 0.9 gm. per kilogram per hour.

It is our intention to study this tolerance in infancy under normal as well as under pathologic conditions. This report deals chiefly with the tolerance of atrophic infants. This class of cases was studied first because it was regarded possible to exercise some beneficial influence by intravenous injections of sugar solutions. The work was done in collaboration with Dr. K. K. Mayer and Dr. P. M. Mattill. A dextrose manufactured by the Corn Products Company marked C. P. was used. It is about 99 per cent. pure. This glucose was dissolved in freshly distilled water. The strength of the solution was made approximately 5, 10, 15 per cent., the actual concentration being determined polariscopically. In making the dilutions we were guided by the fact that with our instrument the most convenient rate of injection was between 25 and 50 c.c. per hour. The syringe takes the solution from a buret, so that the rate of speed can be easily checked. As pointed out before, the strength of the solution is of no consequence, that is,

1. Woodyatt: J. Biol. Chem. **29**:355, 1917.
2. Wilder and Sansum: Arch. Int. Med. **19**:311, 1917.

within wide limits. The limit is determined on one side by the impossibility to inject a very small volume with sufficient accuracy, on the other side by the bulk of fluid which would have to be injected. It is immaterial, as far as the tolerance is concerned, whether 10 gm. glucose are injected in a volume of 10 c.c. or in a volume of 100 c.c., provided it is injected evenly within a given unit of time. Needless to say, apparatus and solutions were properly sterilized. At the start the rate of injection is chosen so that it is not likely to exceed the tolerance. If no sugar appears in the urine in thirty minutes, glycosuria will not occur even after several hours of continuous injection. If no urine is obtained after thirty minutes the time of injection is lengthened until urine can be obtained. In some cases the urine is obtained by catheter, really the preferable method; in others the voiding is remarkably uniform. The urine is tested for sugar with Haines' solution, 1 c.c. of urine sufficing for the test. If the urine does not contain sugar by injecting 0.8 gm. per kilogram per hour, the rate is increased so that 0.9 gm. per kilogram per hour is given, and so on increasing as a rule by 0.1 gm. per kilogram per hour until sugar appears in the urine. If sugar appears at the first rate of injection, the injections are resumed on another day, as it is not possible to work down, at least not without washing out the bladder. If sugar appears at say 1 gm. per kilogram per hour the "tolerance" is taken as 0.9 gm. per kilogram per hour, that is, 0.1 gm. per kilogram per hour lower than where sugar appeared. The injections were usually started two hours after a meal.

We made control injections in five infants that were more nearly of normal weight for their age. Two were cases of tetany and three were inmates of an orphan asylum. These five cases are tabulated in Table 1, and it is seen that the tolerance does not exceed 1.1 gm. per kilogram per hour. In contrast to these findings, the tolerance of the atrophic infants ranges from 1.5 to 1.8 gm. per kilogram per hour (Table 2). The so-called atrophic infants are characterized by emaciation, tendency to subnormal temperature, more or less grayish color of the skin. The weight was stationary or nearly so; the stools were good. We have at present six such cases, a number too small to arrive at any conclusion as to the possible therapeutic value of intravenous glucose injections. As it is, however, in two cases the injections were followed by a gain in weight without change in diet; in one case there was a gain in weight but the diet had also been changed, and in three cases there was no visible beneficial effect.

TABLE 1

No.	Date	Name	Age	Weight	Time	Gm. per Kilogram per Hour	Sugar in Urine
1	4/12	Thad. G.	15	9.09	30	0.8	0
					30	1.0	+
	4/16	do	..	....	30	0.9	+
2	4/20	Tom D.	13½	7.05	30	0.9	0
					30	0.8	0
					30	0.9	0
					30	1.0	+
3	5/16	Sophie	6	4.9	60	0.8	0
	5/23	do	..	....	30	1.2	+
	5/28	do	..	....	30	1.0	+
4	5/22	Norma	5	5.4	30	0.9	0
					30	1.0	+
5	5/17	Earl	7½	5.17	60	0.9	0
					30	1.0	0
	5/19	do	..	....	60	1.1	0
					30	1.2	+
	5/21	do	..	....	30	1.2	+

TABLE 2.—ATROPHY

No.	Date	Name	Age	Weight	Time	Gm. per Kilogram per Hour	Sugar in Urine
1	3/5/19	Stephen K.	6	3.18	30	1.1	0
					30	1.4	0
					30	1.7	+
2	3/31	Chas. S.	7	3.46	30	1.7	0
					30	1.8	+
3	3/24	Viola G.	6	3.2	90	1.7	0
					30	1.9	+
	3/28	do	..	....	60	1.8	+
4	4/18	Francis	5	2.9	30	1.3	0
					30	1.4	0
					60	1.6	+
5	4/19	Mike L.	6	2.9	120	1.4	0
					30	1.6	+
6	4/30	Marie L.	3½	2.5	30	1.0	0
					30	1.1	0
				2.87	60	1.3	0
	5/4	do	..	....	60	1.6	0
				2.87	30	1.8	0
				2.87	30	1.9	+
	5/5	do	..	....	60	1.6	0
					30	1.8	0
					30	1.9	+
	5/7	do	..	3.1	30	1.8	0
					30	1.5	0
					60	1.7	0

We wish to call attention to Viola G., who received 1.7 gm. per kilogram per hour for one and one-half hours without the appearance of sugar in the urine, while 1.8 gm. per kilogram per hour produced

glycosuria. Mike K. tolerated 1.4 gm. per kilogram per hour for two hours, while 1.6 gm. per kilogram per hour gave glycosuria. Mary L. received on three occasions 1.8 gm. per kilogram per hour without the appearance of sugar in the urine, but on two occasions 1.9 gm. per kilogram per hour was followed by glycosuria.

Thus far we have not included blood sugar determinations in our studies, neither did we make any effort at following the blood volume. These features as well as others, especially the tolerance in normal infants, must be left to further study. It would be tempting to bring our results in relation to the known facts, such as the high level of metabolism of atrophic infants as shown by Murlin and Hoobler,[3] with increased temperature of the skin and increased insensible perspiration as shown by McClure and Sauer.[4] As a preliminary, however, it will be necessary to obtain the standards for normal infants at the various periods of life.

Evanston, Ill.

DISCUSSION

DR. SCHLOSS: I would like to ask Dr. Helmholz if in his tests for sugar tolerance in atrophic infants he has repeated the test on successive days.

I have been interested in recording the sugar tolerance in infants, but I have not had the advantage of the Woodyatt method. The method I used was simply to give the sugar with the food. I have examined the blood sugar curve along with the urine with the idea of ascertaining if it might be of some practical value in feeding sugar. The urinary tests were made by the Benedict quantitative method and showed that normal urine contained sugar. The difference between the amount required to reduce copper solution and that in normal urine was perhaps 0.4 gram per 100 c.c. of urine. By increasing the sugar up to a certain point there was always an increase of sugar in the urine, even though it was not enough to reduce copper solutions.

One point of considerable importance is renal permeability. This may vary during an interval of several days. A urine may show a glycosuria of 100 mg. per 100 c.c. on a given amount of sugar and later on glycosuria will occur only when much larger amounts of sugar are given.

DR. MARRIOTT: This paper affords one more piece of evidence that, in considering the metabolism of the infant, it is the active protoplasmic mass rather than the whole body weight which has to be considered. The fat is merely an overcoat, and the atrophic infant without the overcoat of fat needs and can utilize in the intermediary metabolism almost as much food in the day as a normal infant of the same age and length, as he has about the same amount of active protoplasmic tissue. In the case of atrophic infants we have observed the same high tolerance to carbohydrates when administered by mouth as Dr. Helmholz has when administered intravenously. In the case of a very atrophic

3. Murlin and Hoobler: Am. J. Dis. Child. **9**:81, 1915.

4. McClure and Sauer: Am. J. Dis. Child. **10**:425, 1915; Arch. Int. Med. **21**:428, 1918.

infant sugar first appeared in the urine after the administration of a daily amount of sugar corresponding to 1.5 gm. per kilogram per hour, a figure quite comparable to those of Dr. Helmholz.

DR. HELMHOLZ: In answer to Dr. Schloss' question, our results have been remarkably constant when the determinations were made at intervals of two or three days.

In regard to the glycosuria, Woodyatt has shown that if an individual does not secrete sugar in the urine after a half hour, at a certain rate of injection, you can keep on giving that amount indefinitely without producing glycosuria. This has been pretty definitely proved.

A CASE OF HODGKIN'S DISEASE IN A GIRL OF TWO YEARS

LANGLEY PORTER, M.D., M.R.C.S. (Eng.)
SAN FRANCISCO

The morbid entity known as Hodgkin's disease or lymphadenoma, is not infrequently found in children. For the most part, however, those attacked are in their second decade. Hugh Thursfield says that of a hundred admissions for this disease to Bartholomew's Hospital for ten years, thirty were patients less than ten years of age. During the same time thirteen undoubted cases were admitted to the wards of the Great Ormond Street Hospital for Children, an admission percentage of 0.1 per cent. All reporters emphasize the fact that the disease is very rarely encountered in children under 5 years of age, and it is practically unknown in infancy. Reginald Miller records the youngest authentic instance of the disease, the child being 25 months of age at the onset. The child we are reporting was 30 months when she came under observation; the onset of her disease apparently came at her 27th month.

The unusual features that make this case worth reporting are: (1) the early age of the onset; (2) the female sex of the child (males are affected at a ratio of 6:1); (3) the rapid onset which followed what was undoubtedly an infection involving the gastro-intestinal tract; (4) the early and extreme involvement of the mesenteric and retro-peritoneal glands; (5) the late and slight involvement of the cervical glands, which are usually affected early and profoundly; (6) the remarkable, extensive, and early involvement of the skin in morbid process; (7) the extreme edema of the legs while the anemia was still far from profound; (8) the intermittence of the fever and the toxic symptoms, which on several occasions gave an unwarranted hope for improvement; (9) the repeated but temporary improvement of the child after blood transfusions; (10 the absence of any demonstrable infective agent — *Cornebacterium hodgkini*, or other organism in the glands removed for diagnostic purposes; (11) the extensive invasion of the lung septa by the endothelial cells characteristic of this disease (ordinarily such invasion is limited to the peribronchial and superficial pleural areas of the lung); (12) the unusual size of the spleen, which in this disease most often is but moderately enlarged.

The difficulties of diagnosis in this case were largely matters of reaching a decision between lymphadenoma and lymphosarcoma. The absence of early jaundice, the small liver, the huge spleen, the lack of any tendency of the glands to infiltrate, and the protracted course finally forced opinion to the diagnosis of lymphadenoma, a view that was amply confirmed by studies of tissue sections.

This case unfortunately brought only more evidence to the sad array which proves that up to now we have discovered no therapeutic resource of avail in the amelioration of lymphadenoma. There is nothing that we can turn to with any hope of aiding these patients; removal of focal infections, autogenous vaccines, hygienic measures, blood transfusions, roentgen-ray exposures, radium emanations, arsenic in any form, all fail when put to the test.

CASE REPORT

L. B., a girl, aged 2½ years, was first seen in March, 1917. The complaint was that the child was suffering from a fever, anorexia, anemia, and profound malaise of increasing intensity.

The family history was without importance, and up to March, 1917, the child had been in perfect health. At that time a severe dysentery attacked her as well as a number of other children, her playmates. This attack was attributed to the fact that she had eaten spoiled apples. A few weeks before this she had had a retropharyngeal abscess which had been opened and which healed without incident. Late in May she developed a persistent but not severe cough. There was occasional vomiting. Her temperature had risen almost every day. She suffered from marked flatulence, no abdominal pain, a capricious appetite, disturbed sleep, increased pallor and an intense itching of the skin.

At admission to the hospital her weight was 23 pounds. She was an emaciated, pale child, giving evidence of much discomfort. The pupils were dilated, otherwise eyes, ears and teeth showed no abnormalities. The tonsils were enlarged and red, all visible mucous membranes were pale. There were a few small anterior and posterior cervical glands palpable and visible. The chest was thin, moved equally and fairly well. There was diminution in the resonance of the right lung anteriorly and at the right apex behind. The breath sounds were exaggerated; breathing was very high pitched on the right of the spinal column between the third and sixth dorsal. Scattered râles were heard, especially at the base of the right lung. There was dulness over the spine to the fourth dorsal and whispered pectoriloquy to the same point. There was nothing abnormal about the heart except that there was a diastolic murmur audible over the whole precordium, loudest at the third left space. There were no other murmurs.

The abdomen was very much distended, spleen was extremely large and very hard. It extended as far forward as the umbilicus and down to within an inch or less of the iliac crest. Masses of large, hard glands could be palpated everywhere throughout the abdomen and the liver was not unduly large. The

shifting dulness of both flanks was undoubtedly due to presence of fluid. The extremities were thin with marked edema of both feet and legs half way to the knees. The genitalia were normal.

The skin everywhere, more especially the skin of the body, was covered with scattered, slightly raised lenticular papules, varying in size from a pinhead to a small navy bean. These were pale in color, and apparently caused intense itching for the skin was extensively marred with scratch marks.

During six weeks' stay in the hospital the child gained a pound, improved in every way, the spleen decreased very materially in size and during this time her blood improved. On admission the blood showed a hemoglobin of 35 per cent., number of red blood corpuscles 2,290,000, white blood cells 20,000, neutrophils 82 per cent., eosinophils 1 per cent. At the end of six weeks there was a gain of 20 per cent. in the hemoglobin and a million and a half in the red cells. The Wassermann and urine were negative. Nose and throat cultures showed staphylococci. The temperature during the first week ran steadily to 102 F., and gradually fell to normal. Treatment during this time was chiefly dietetic, reenforced by injections of iron cacodylate. She left the hospital and continued on with the hygienic treatment.

She had a relapse and went steadily down hill and again came under our observation in October, 1917. Her hemoglobin at this time was 40 per cent., red cells 3,280,000, with normal white and differential. She was transfused and following the transfusion her temperature went to 103 F., but came to normal the next day, and there was a slight temporary improvement in all symptoms, especially in well-being and appetite and the edema was completely cleared up and did not return. On October 4 she was transfused once more, and again on November 1.

After dismissal from her second admission the blood was studied by Samuel Hurwitz and on several examinations at two or three day intervals was found to be normal except for the low hemoglobin and a moderate diminution in red cells. The deformity of the red cells and polychromasia were trivial.

BLOOD EXAMINATIONS

Sept. 22, 1917: Hemoglobin (Dare), 27 per cent.; red blood corpuscles, 3,008,000; white blood corpuscles, 9,000. Smears: Red blood cells extremely pale; only moderate anisopoikilocytosis. No nucleated red blood corpuscles. The polymorphonuclears constituted 72 per cent. of the total leukocytes. Platelets abundant.

September 27: Transfusion at 2 p. m. Blood examination at 5 p. m. Hemoglobin, 40 per cent.; red blood corpuscles, 3,280,000; white blood corpuscles, 12,000.

Oct. 4, 1917: Test for the fragility of the red blood corpuscles. Maximal resistance (average normal, 4.7) 5. Minimal resistance 3.5, normal.

October 9: Hemoglobin, 28 per cent.; red blood corpuscles, 2,576,000; white blood corpuscles, 6,000. Smears: Red blood corpuscles, pale; centers clear; some distortion in size and shape. One nucleated red corpuscle seen.

October 13: Hemoglobin, 35 per cent.; red blood corpuscles, 2,736,000; white blood corpuscles, 4,000. Smears: Moderate anisopoikilocytosis. Some polychromasia. Polymorphonuclears constituted 68 per cent. of the white cells.

On Jan. 23, 1918, after having been in fair health for some weeks she was taken suddenly with clinical signs of pneumonia, together with an enormous dilatation of the heart. Moderate enlargement of the posterior cervical and submaxillary glands had now appeared for the first time. The breathing was

urgent and rapid, pulse weak and irregular. The breathing was aggravated by the enormous distention of the abdomen which on palpation showed the exaggerated state of the physical condition as previously described. She died at 4 a. m., January 24, and a necropsy was performed by William Ophuls, assisted by Dr. Anna Macrae. I am much indebted to Dr. Ophuls for a very painstaking study of the tissues, his necropsy report and microscopic findings which follow.

NECROPSY

Fairly well built, rather poorly nourished girl of about 2 years.

Skin and visible mucous membranes pale, teeth, normal.

Large masses of rather hard, fairly discrete glands in the posterior part of the neck, and in the submaxillary region on both sides. On left larger than on right.

On palms of both hands, some rough places, especially at sides of palms.

Axillary glands are large and hard (size of lima bean).

Skin of trunk is quite rough, full of small, hard nodules, from just visible to 2 or 3 mm. in diameter. Some of these are covered with scabs, others show like little white scars.

Small petechial patches on both legs in vicinity of knees.

Almost complete absence of subcutaneous fat. Marked atrophy of muscle.

Peritoneum and abdominal muscles very pale.

Lung shows a diffuse, grayish red consolidation posteriorly, involving practically entire lung.

Pleura on both sides show slight diffuse thickening, with a slight hemorrhage. Bronchial glands, right same as left. Right lung shows some diffuse consolidation except for anterior part of middle lobe. Bronchi on both sides are filled with mucopurulent material.

Heart, enlarged in diastole. P. M. slow in pulmonary artery. Heart muscles pale and flabby. Valves, aortic, pulmonary, coronaries, normal.

Aorta surrounded by enlarged glands; considerably compressed but not invaded. The vena cava and iliac veins are also somewhat compressed, but otherwise free.

Blood in large vessels very thin and watery. In the peritoneal cavity there is about 50 c.c. of clear, yellow fluid.

Both pleural cavities contain a small amount of bloody fluid; about 250 c.c. of clear fluid in pericardium.

Spleen much enlarged, projecting four finger breadths below the free margin of the ribs. Some adhesions between spleen and omentum. Capsule shows a slight irregular thickening. There are a few pitted scars on convexity. Measures 5½ by 3 by 2 inches. Very firm. Cuts with difficulty. Cut surface is of dark brown color, full of just visible grayish nodules.

Liver projects three finger breadths on the right side and disappears behind ribs in midline on left side. Measures 8 by 5 by 3 inches. Capsule shows slight diffuse thickening. Liver tissue firm, pale, grayish red, full of minute yellow spots. Bile duct patent.

Stomach and transverse colon pushed down. Stomach contains some mucus and partly digested milk. Mucous membrane very pale, otherwise normal.

Abdomen very much distended, also lower part of the chest. Several glands about size of hazelnut in inguinal and femoral region, where there are some scars.

Omentum very thin and shows a few small reddish glands.

In left gastroceliac region there are many grayish-red, hard, enlarged glands; largest is the size of a hazelnut.

Diaphragm at fourth rib on both sides. In anterior mediastinum is small remnant of thymus and several large hard glands.

Retroperitoneal lymph nodes are all markedly enlarged and the tissue about them is very hard and indurated. The glands themselves show a brownish cut surface. Bone marrow very soft, of a reddish brown color.

Left adrenal buried in dense fibrous tissue, shows marked atrophy, otherwise normal.

Left kidney somewhat swollen; pale; measures 3½ by 1 by 2 inches. Cut surface shows marked edema.

Right adrenal same as left.

Right kidney about half size of left; somewhat more congested; does not show so much edema.

Pelvic organs normal, except for large masses of glands along the iliac arteries.

All the mesenteric glands are greatly enlarged; largest, size of a walnut.

Pancreas tissue shows some large glands at tail and head; also enlarged glands at hilus of liver.

Gallbladder contains some thin bile. Appendix normal.

Intestines normal; no sign of lymphoid hypertrophy.

Cervical glands same as others.

Esophagus and thyroid large. Trachea normal.

Diagnosis. — Lymphadenoma, general. Hydropericardium. Bronchopneumonia. Hodgkin's disease in child involving lung.

MICROSCOPICAL EXAMINATION

Sections of spleen show irregular masses of dense fibrous tissue about the small arteries. There is a good deal of pigment in the tissue; besides this, there are numerous small, nodular areas made up of large fusiform and star-shaped nodules. Large, light nucleated giant cells, no evidence of cells with large light nuclei. There are some large, multinucleated giant cells, no evidence of caseation. Many of the cells are heavily pigmented. No parasites found in any of these giant cells.

Sections of lymph nodes show small remnants of lymphadenoid tissue; otherwise irregular masses of dried nodules of the kind described in the spleen.

Sections of the skin show in the papillary layer groups of large cells very much like those that form the nodules in the spleen.

Another lymph gland shows a filling of the lymph spaces with these large mononuclear and giant cells.

Lung shows a diffuse cellular thickening of the septa causing a marked narrowing of the spaces. The thickened septa contain lymphocytes and large mononuclear cells. The air spaces are very much narrowed, lined with proliferated epithelium; some of them are filled with masses of degenerating cells.

Certain of the lymph glands show the structure of fibrosarcoma with large fusiform cells. Some of the newly formed tissues in lymph glands show marked fatty degeneration.

Pancreas shows a marked fibrous thickening of the interlobular connective tissue.

Liver shows marked dilatation of the capillaries, the dilated capillaries being filled with blood in which there are many leukocytes and some large mononuclear cells.

In periportal connective tissue there are several nodules made up of large cells.

Kidney shows a marked congestion and edema; tubules are wide and filled with granular material. Glomeruli are rather large and cellular.

SMEARS

Bone marrow shows some neutrophilic myelocytes, many neutrophilic leukocytes, many large irregular cells with a somewhat spongy protoplasm that stains blue with Giemsa method. Some of the cells are pigmented; very few eosinophils. The red blood corpuscles are of irregular sizes and shapes. Few nucleated red blood corpuscles; few short chains of streptococci. Some multinucleated giant cells.

The spleen shows many of the large cells with the blue protoplasm; few neutrophilic nucleated red blood corpuscles. No cocci found.

240 Stockton Street.

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